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ARCHIVES OF SURGERY

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SYMMETRICAL TRAUMATIC FRACTURES OF THE CRANIUM; SYMMETRICAL FRAGMENTATION

COMMENTS ON THEIR MECHANISM

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AND

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CHICAGO

During thirteen and a quarter years, one of us (E. R. L.) made or took part in 1,278 postmortem examinations of the bodies of persons who at the time of death had fresh or recent traumatic linear fractures of the cranium such as usually are produced by blunt force. Fractures due to bullets or other missiles are not included in this group, nor are cleancut sharply localized perforating wounds of the cranium due to cutting implements or weapons. Twenty-six of the 1,278 fractures were in persons not more than 1 year old; 45, in persons from 1 to 10 years of age and 29, in persons between the ages of 11 and 20 years. This proportion of fractures in adults, 1,178 of the 1,278, is about the same as Vance [1] found in 507 similar fractures, with 66 in persons in the first two decades of life. From time to time, publications have issued from different clinics and hospitals based on the clinical and anatomic observations on fractured skulls during certain periods.[2] It is interesting to find in examining such studies that the features of cranial fractures encountered a few decades ago are different from those now prevalent. In the large series analyzed by Vance,[1] as well as in those furnishing material for our study, long radiating fractures are numerous. A number of years ago, localized fractures with comminution and depression were relatively more common. This change is undoubtedly a result of the increase in accidents from motor vehicles.

During our postmortem examinations, the course of the fractures was carefully drawn on printed charts. As a routine measure the

From the Norman Bridge Pathological Laboratory of Rush Medical College.

1. Vance, B. M.: Fracture of the Skull: Complications and Causes of Death; A Review of Five Hundred and Twelve Necropsies and of Sixty-One Cases Studied Clinically, Arch. Surg. **14**:1923 (May) 1927.

2. (*a*) Dwight, E. W.: A Study of One Hundred and Sixty-Nine Autopsies in Which Fractures of the Skull Were Found, M. & S. Rep. Boston City Hosp., 1894, p. 129. (*b*) Brun, H.: Der Schädelverletzte und seine Schicksale, Beitr. z. klin. Chir. **38**:192, 1903.

diagrams thus made were compared with the fractures by a second, and often a third, person to confirm their accuracy. Tension was made to spread open the fractures, fragments being pulled apart slightly to determine the limits and connections of the fissures. In examining these sketches recently, it was found that some of the fractures were distributed equally on the right and left sides of the skull, or that fractures coursing in the sagittal midline had broken the cranium in lateral halves. The resulting symmetry of fractures and fragments is by no means perfect in the 80 selected from the 1,278 fractures because of such characteristics.[3] In a few, the symmetry is beautiful, but in most of the 80 there are some deviations or irregularities; however, there are not enough for their inclusion with the more perfect fractures to lessen their usefulness in this study.

A great deal of attention has been devoted to the way the skull breaks from external blunt violence.[4] Many of the investigations have been experimental. The desire to compare accepted views regarding the mechanism of linear skull fractures with the information available concerning the 80 fractures was both natural and resistless. Experiments have failed to reproduce similar symmetrical fractures with any regularity. The symmetry or approach to symmetry in the 80 fractures suggested a greater likelihood of obtaining notions from them of how craniums break than from a study of the usual run of skull fractures from which they were selected. Almost one half of the 80 are ring-shaped fractures about the foramen magnum, mainly in the posterior fossa, or spread out so as to include the posterior fossa and also portions of the middle fossa and the vault.

We have not attempted to fix the time definitely when the casual relationship of the spine and the weight of the body and its extremities to ring fractures about the foramen magnum was first fully understood. New ideas are often announced independently from several sources and sometimes at intervals of many years before they are integrated and conventionalized. So far as French literature is concerned, there is some indication that the explanation by Berchon[5] in 1862 of a ring fracture of the base of the skull was novel. The

3. Symmetrical fractures of the mandible, of other bones of the face and of the zygomatic arches have been omitted.

4. Two other articles on skull fractures based on some of this series of 1,278 fractures have been published: (*a*) Le Count, E. R., and Apfelbach, C. W.: Pathologic Anatomy of Traumatic Fractures of Cranial Bones, J. A. M. A. **74**: 501 (Feb. 21) 1920. (*b*) Apfelbach, C. W.: Studies in Traumatic Fractures of the Cranial Bones, Arch. Surg. **4**:434 (March) 1922.

5. Berchon, E.: Fracture communicative du frontal avec enforcement des fragments et irradiations nombreuses et étendues, Bull. Soc. anat. de Paris, series 2, **7**:80, 1862.

fracture occurred in a man who fell into the hold of a ship that was under construction (fig. 1). Berchon made a complete break from traditional explanations of fracture by contrecoup and said that the basilar fracture was direct and produced by the shock of the cervical vertebrae charged with all the weight of the body. Trélat, in discussing Berchon's report, frankly said that this was a new way of accounting for such fractures. Previous to this time, Trélat had made two noteworthy contributions to the subject of skull fractures.[6] He also had described a ring fracture in the posterior fossa in a report on a necropsy which was added to a clinical account by Tarnier.[7] In this case, a man, aged 50, had fallen headlong out of a window onto a paved court and died at once. Interest in his injuries apparently

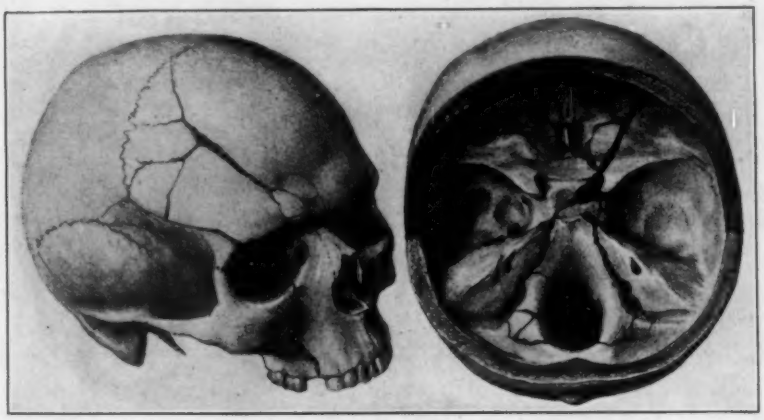

Fig. 1.—The ring fracture reported by Berchon in 1862. (From Berchon: Bull. Soc. Anat., series 2, **7**:80, 1862.)

was mainly in a torn articular cartilage of the left knee, for Broca, Fano, Verneuil and Bourcy discussed Tarnier's presentation without mentioning the cranial fracture. Although Trélat said, in describing the fracture, "Il résulte de la multiplicité de ces lésions et de la direction des lignes suivies par elles, que la partie centrale de la base du crâne formait une sorte d'anneau irrégulier complètement indépendant du rest du crâne et reste seul en connection avec la colonne vertébralé," he said nothing as to the actual method of its production. But when Trélat heard Berchon's report seven years later, he recalled

6. Trélat, U.: (*a*) Mémoire pour les prix de l'Internat, 1852; (*b*) Des conditions de résistance du crâne, Bull. Soc. anat. de Paris, series 1, **30**:121, 1855.

7. Tarnier: Décollement traumatique d'un cartilage; fracture du crâne, Bull. Soc. anat. de Paris, series 1, **30**:193, 1855.

the fractured skull in Tarnier's patient and supplied an illustration to accompany that of the ring fracture described by Berchon (fig. 2). E. Simon, secretary for the society when these announcements and discussions took place, paid high tribute to Berchon in his review of the work for 1862, thanked him for the society and said that they were honored by having such an important matter presented to them.[8]

This account is remarkable, because Denucé,[9] in discussing the paper by Trélat in 1855, criticized him severely for failure to emphasize the influence of the spine. Among other statements by Denucé is this:

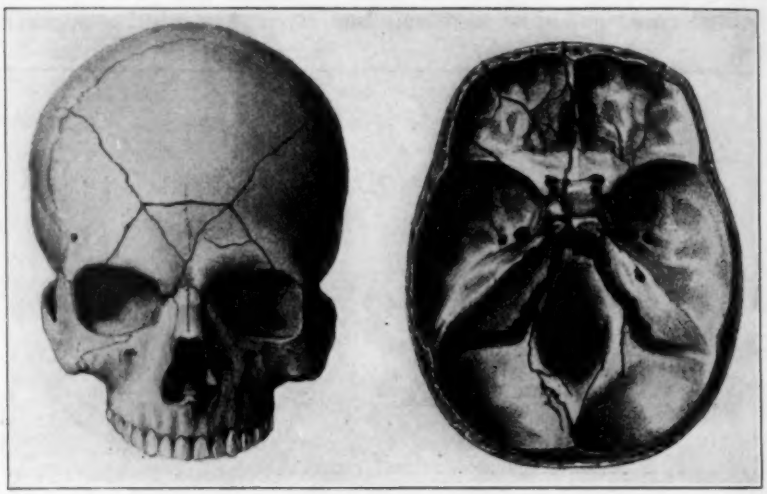

Fig. 2.—The ring fracture reported by Trélat in 1855. (From Trélat: Bull. Soc. Anat., series 1, **30**:121, 1855.)

Enfin un dernier reproche que je ferai à M. Trélat, c'est d'avoir négligé complètement les conditions de résistance que peut offrir le crâne lorsque le choc vient de bas en haut, et dans tous les cas, l'action que doit exercer le poids du corps par l'intermédiaire de la colonne vertébrale sur la base du crâne, alors même, comme cela se présente dans beaucoup de chutes, que le choc principal atteint la voûte.

It is altogether likely that when Berchon's presentation was made, Trélat was fully aware of the action of the spine and the weight of the trunk, but had not connected them with ring fractures about the foramen magnum. It is well to recall that when these events occurred almost a century ago, controversy was still active regarding isolated fractures distant from the place injured: contrecoup fractures, as they were

8. Simon, E.: Bull. Soc. anat. de Paris, series 2, **7**:534, 1862.

9. Denucé, in discussion on Trélat,[6b] p. 132.

called. The polemic, beginning with the time of Hippocrates,[10] had its first period of merely differences of opinion concluded by the efforts of the French Academy of Surgery to secure a satisfactory answer to the problems of such fractures by offering prizes in 1760, 1765 and 1768.[11] Among the awards was one to Saucerotte for his vibration theory. Briefly stated, this predicated a disrupting ebullition of vibrations where they came together at the opposite pole after traveling around the skull in various meridians from the place struck.

During the second period, which ended with the publication of studies by Aran,[12] the theory of Saucerotte dominated, and these fractures received relatively little attention. It is of interest that Aran called them independent fractures. Since his announcement of the paths taken when fractures radiate from the vault to the base of the skull, there have been no definite periods of discussion. Schroen,[13] referring probably to the measurements of cranial elasticity by Bruns,[14] said:

> Deutschen Forschern gelang es, die rechte Fährte zu finden; schon schien es als ob das Ziel diesmal in Wirklichkeit erreicht würde, schon reifte die Frucht der suchenden Hand entgegen, als abermals von Frankreich eine neue fascinierende Theorie die deutschen Arbeiten zum Stocken brachte, es war die Felicet'schen Hypothese,[15] die man im Gegensatze zu der ersten physicalischen französischen Theorie die "anatomische" nennen könnte.

This anatomic period of Aran and Félizet, with many embellishments from physics, still prevails. But the Saucerotte vibration theory died out slowly, and for many years any observation of an independent fracture of the cranial base was likely to find its way into literature. Alone or associated with other fractures, and regardless of their loca-

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10. Many accounts of the development of the present knowledge of the mechanism of these independent cranial fractures have been written. The following are perhaps the most complete: (*a*) Friedberg, H.: Klinische und forensische Beiträge zu der Lehre von den Kopfverletzungen, Virchows Arch. f. path. Anat. **22:** 39, 1861. (*b*) Dulles, C. W.: The Mechanism of Indirect Fractures of the Skull, Tr. Coll. Phys., Philadelphia **8:**273, 1886. (*c*) Schroen, F. W. C. A.: Historisches und Theoretisches zu der Lehre von den Schädelbrüchen, speziell den sog. Gegenbrüchen, Inaug. Dissert., Würzburg, C. J. Becker, 1902.

11. Mémoire sur le sujet proposé pour le prix de l'académie royale de chirurgie, Paris, 1778, vol. 4.

12. Aran, F. A.: Recherches sur les fractures de la base du crâne, Arch. gén. de méd., series 4, **6:**180 and 309, 1844.

13. Schroen,[10c] p. 4.

14. von Bruns, V.: Handbuch der praktischen Chirurgie, Tübingen, H. Laupp, 1854, p. 203.

15. Félizet, G. M.: Recherches anatomiques et expérimentales sur les fractures du crâne, Paris, A. Delahage, 1873.

tion, they are uncommon. Deroubaix,[16] however, said that he often saw them in his own postmortem work and regretted his failure to keep accurate records of them. In discussing fractures of the base of the skull in relation to incidence, most writers, probably influenced by the work of Aran,[12] have been content to state the number of fractures radiating into this or that fossa from the vault, and how many as well as which fossae were involved, when the fracture, or fractures of both the base and the vault, coursed through more than one fossa. But few writers have recorded how many fractures in their series were strictly limited to the base of the skull. Schwartz[17] found 7 among the 115 cases which he analyzed; Walton,[18] 7 among 50, and Phelps,[19] in three contributions, mentioned 4 in 45, 17 in 126 and 15 in 312, respectively.

The infrequency of fractures confined to the base of the skull has not been the sole reason for the many reports concerning them. Advantage has been taken of the opportunity offered by such fractures to repeat refutation of their origin by contrecoup vibration. In 1842, Guthrie[20] said:

> The late Mr. Earle supposed that a fracture of the base of the cranium depended on the occiput being forcibly impelled against the atlas. Sir Charles Bell has maintained the same opinion, and given a scientific account of the manner in which the mischief takes place. It appears to me that this accident principally depends on the superincumbent weight of the body pressing on the unsupported flat, and thin base of the skull, and is but little connected with the unyielding nature of the spine; for it occurs to as great an extent in consequence of falls from a short distance without any impetus, as from falls from a great height.

A number of years before, the opinions of Mr. Earle were also stated by Brodie,[21] who wrote, regarding contrecoup fracture:

> It has been observed to me, however, by Mr. Earle, that he has not known a fracture of this kind to take place, except where the blow seems to have operated

16. Deroubaix: De la lésion de certain nerfs, considérée comme moyen de diagnostic des fractures de la base du crâne, Bull. Acad. roy. de méd. de Belgique, series 4, **4**:740, 1890.

17. Schwartz, A.: Zur Statistic der Fracturen der Schädelbasis, Inaug. Dissert., Dorpat, 1872.

18. Walton, G. L.: Fracture of the Base of the Skull, Ann. Surg. **40**:654, 1904.

19. Phelps, C.: (*a*) A Clinico-Pathological Study of the Injuries of the Head, New York M. J. **57**:29, 1893; (*b*) Traumatic Injuries of the Brain and Its Membranes, New York, D. Appleton, & Company, 1897; (*c*) Analytical and Statistical Review of One Thousand Cases of Head Injury, Ann. Surg. **49**:449, 1909.

20. Guthrie, G. J.: On Injuries of the Head Affecting the Brain, London, J. Churchill, 1847, p. 65.

21. Brodie, B. C.: Pathological and Surgical Observations Relating to Injuries of the Brain, Med.-Chir. Tr., London **14**:325, 1828.

in such a manner as to impel the occiput forcibly against the atlas, the line of fracture passing through the former bone where it rests on the latter.

These comments are not concerned directly with ring fractures about the foramen magnum. Ring fractures acquired whatever they now possess of entity and comprehension only gradually and during the years when, as already stated, case reports of all sorts of independent fractures of the base of the skull were customary.

In some skulls, basal fractures are the only ones present. For these, we believe that the term solitary basal fracture is most suitable. We prefer to restrict the name independent to fractures associated, but not continuous, with the main fractures located at, and radiating from, the site of violence. Many writers call them indirect, whether they are solitary or not. By many, the terms contrecoup and indirect have been used synonymously; even in recent years, these fractures have occasionally been labeled contrecoup.

Robert, in 1843,[22] reported fractured posterior clinoid processes associated with a fracture across the right petrous bone, the skull bones being otherwise intact. Robert thought that the fracture in the petrous bone was contrecoup, but Aran said regarding it:

> Nous ne voulons pas disputer sur ce fait, nous l'acceptons tel qu'il est; mais nous ne pouvons voir là une fracture par contre-coup. Qu-importe, en effet, que la puissance exerce son action d'une manière immédiate sur la base du crâne ou qu'elle y arrive par l'intermédiaire de la colonne vertébrale?

The body was that of a mason who had fallen 12 meters, striking feet first. Announcements of similar fractures were so few at this time that von Bruns,[23] a decade later, mentioned only one other,[24] with that reported by Robert, as examples of basal fractures due to transmission of violence by the spine. The report by Marmy, concerned fractures also due to alighting feet first; in the cranium the ethmoid cribriform plate was broken by a solitary fracture. Another report of such fractures of the base by Marmy, which Bruns should have included,[25] dealt with

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22. Robert: L'expérience, J. de méd. et chir. pract., Nov., 1843; as cited by Aran, and many others. These fractures are illustrated by Victor von Bruns in Chirurgischer Atlas. Bildliche Darstellung der chirurgischen Krankheiten und der zu ihrer Heilung erforderlichen Instrumente, Bandagen und Operationen, Tübingen, H. Laupp, 1853, fig. 22, plate IV.

23. von Bruns,[14] p. 281.

24. Marmy: Fractures multiples (du calcanéum, du crâne, et du radius), Bull. Soc. anat. de Paris, series 1, **23**:258, 1848.

25. Marmy: Fractures du crâne et déchirure du poumon et du foie. Epanchement du sang dans l'abdomen; fracture du poignet, Bull. Soc. anat. de Paris, series 1, **23**:193, 1848. Both reports by Marmy are cited by Denonvilliers and Gosselin (Compendium de chirurgie pratique, Paris, 1851, p. 580) as examples of fractures caused by blows of the spine from below, as they were subsequently cited by E. von Bergmann (Die Lehre von den Kopfverletzungen, in Deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1880, pt. 30, p. 208).

fractures in the right petrous bone and body of the sphenoid in a man who fell from a second story window and alighted on his hands and knees. Bruns [26] emphasized the manner in which the skull is compressed vertically by the weight of the oncoming trunk after the head is arrested, but he did not mention ring fractures.

If it is true, as Guthrie said, that Bell understood "how the mischief takes place," he may indeed have been the first to observe a ring fracture of the base of the skull and at the same time the first to comprehend its mechanism. He described a fracture [27] that "began by the side of the petrous bone of one side, and extended around the occipital bone behind the foramen magnum to the petrous portion of the temporal bone of the opposite side." It was caused by a bucket which fell 50 feet and hit the back of the head of a man in a tunnel. As will be shown presently, this is an important observation. Bell went on to say that the blow was received "on the strongest part of the skull, in the convexity of the occipital bone; the fissure was in the weakest part." The fracture was an incomplete ring with the gap in front, and belongs to the type we are discussing. But it is not altogether evident that Bell was fully aware that resistance from below assisted in producing the fracture, for he concluded with: "It was a pure instance of counterfissure."

Forgues [28] mentioned symmetrical fractures described by Couderc,[29] one on each side from the lambdoid suture to the jugular foramens. Such fractures are portions of a ring.

In 1858, Hewett [30] described a similar incomplete ring fracture due to a fall of about 20 feet (6 meters); the scalp wound was on the right side of the vertex behind. In the body of a man 32 years old, who fell off a scaffolding, Bryant [31] found that "the weight of the body, as conveyed through the spine, had completely driven the spinal column into the skull, the base for about one inch round the foramen magnum being detached and pressed inwards upon the brain; this was much lacerated and contused from the pressure of the displaced bone; fissures radiated upwards from this spot." The cranial vault was uninjured. Accompanying fracture of both articular processes of the epistropheus,

26. Bruns,[14] p. 285.

27. Bell, C.: Surgical Observations, London, Longmans, 1816, p. 467.

28. Forgues, L. D.: Mécanisme des fractures du crâne, Thèse de Strasbourg, 1869, p. 30.

29. Couderc, J. A.: Sur les fractures du crâne, Thèse de Paris, 1850.

30. Hewett, P.: Lectures on the Anatomy, Injuries and Diseases of the Head, M. Times & Gaz. **16**:27, 1858.

31. Bryant, T.: The Injuries and Diseases of the Nervous System, Guy's Hosp. Rep. **20**:59, 1859; case 36.

Gurlt [32] found a piece broken from the rim of the foramen magnum. The injuries were in the body of a man 24 years old, who fell head first from a wagon and struck on the crown of his head. Discussion of this fracture by von Bergmann a few years later [33] leaves the impression that the piece broken off was from considerable of the circumference of the margin of the foramen. The large ring fracture described by Chauvel [34] was elliptic. Its posterior end was formed by the foramen magnum. Branches beginning on each side behind the occipital condyles forked symmetrically, with short arms ending in the lower occipital fossae and long arms extending forward through each petrous bone and each greater wing of the sphenoid to meet just behind the crista galli. Chauvel's thesis contained no illustrations, but Chipault and Braquehaye [35] showed the path of this ring in a diagram of the outside of the base of the skull. The injuries were due to a fall of 2.5 meters so as to alight on the head; the scalp wound was in the right parietal region, and the vault was not broken. Both Félizet [36] and Forgues [37] dealt with Chauvel's fracture at length. Inspired by his examination of this ring fracture, Chauvel attempted to make a similar fracture experimentally. In 1 skull he obtained rather symmetrical fractures, 3 cm. long on one side and 5 cm. long on the other, running back from each jugular foramen into the inferior occipital fossae. The body was that of a hunch-backed man, 73 years old, with the first 6 vertebrae ankylosed. Chipault and Braquehaye [38] also illustrated the injuries in this skull.

Some indication of the attention these ring fractures received at this time is evident in the circumstantial description given of one of them by Hutchinson.[39] One of the two fine illustrations accompanying his report was reproduced by von Bergmann.[40] Hutchinson said, "it is very unusual in practice to find the line of fracture symmetrical, probably because in but few instances is the direction of the violence exactly

32. Gurlt, E.: Handbuch der Lehre von den Knochenbrüchen, Hamm, G. Grote, 1862, p. 35.

33. von Bergmann: Verletzungen und chirurgische Krankheiten des Kopfes, in Pitha and Billroth: Handbuch der allgemeinen und speciellen Chirurgie, Stuttgart, Ferdinand Enke, 1873, vol. 3, p. 129.

34. Chauvel, F.: Essai sur les fractures du crâne, Thèse de Paris, 1864, p. 66.

35. Chipault, A., and Braquehaye, J.: Etudes graphiques sur les fractures indirectes de la base du crâne (définition et mécanisme), Arch. gén. de méd., series 8, **4**:279, 394 and 665, 1895, fig. 22, p. 292.

36. Félizet,[15] p. 145.

37. Forgues,[28] p. 12.

38. Chipault and Braquehaye,[35] fig. 23, p. 292.

39. Hutchinson, J.: Circular Fracture of the Base of the Skull with Features of Unusual Interest, Tr. Path. Soc. London **17**:254, 1866.

40. von Bergmann,[33] p. 130.

vertical." The basilar process, occipital condyles, body of the sphenoid and inner halves of the petrous bones made "one nearly circular fragment." A fissure from the ring ran up in the vault from the right middle fossa. The fracture was due to a headlong fall, and Hutchinson understood the part played by the impact of the spine and the weight of the trunk in its production. Schwartz [41] failed to state where he found the elliptic fracture of the base which he said encircled the foramen magnum, sella turcica and crista galli. The particulars of its course are similar to those of the elliptic ring fracture described by Chauvel.[34] The review by Schwartz comprised fractures in 115 skulls; 14 from the Dorpat collection, 25 from Obuchow and 75 from the literature. Details of the course of the fractures were meager in about one half of the 115 cases. Only 7 were limited to the base, and only 1 was a ring fracture.

Félizet [42] broke out a large circular piece from the base of the skull experimentally without fracturing the vault. It was nearly all ventral to the foramen magnum and included the sella and a large part of each petrous bone. He did this by hitting the padded vertex with a large hammer. The support for the base was patterned after the spine. The following is an account by Bennett [43] of the injuries he found in the cranium and spine of a man hit on the head by a weight which fell 34 feet (10 meters).

> . . . having been checked in the fall at a distance of nine feet above the ground. . . . There was an ordinary depressed fracture of the parietal bone, and a fracture by radiation was marked by a fissure extending from the depression to the orbital plate of the sphenoid bone, and passing into both optic foramina. . . . The base of the cranium was fractured over each occipital condyle, and an oblique fracture passed outward from the foramen magnum to end in the petrosal sinus. The transverse process of the first cervical vertebra was detached, the spine had been driven upward and was actually impacted into the cavity of the cranium. The posterior clinoid processes were also detached.

The scalp wound was in the left parietal region. Symmetrical fractures from each jugular foramen to about 1 cm. behind the occipital condyles, associated with detached posterior clinoid processes, fracture of the body of the sphenoid bone and loosened sutures between the occipital and the temporal and parietal bones on the right side, were reported by Magon.[44] They were in the head of a man 43 years old who fell backward and head first from a ladder.

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41. Schwartz,[17] p. 19, case 4.
42. Félizet,[15] p. 146, plate 13.
43. Bennett: Fractures of the Cranium, Brit. M. J. **1**:326, 1875.
44. Magon, E.: Fractures multiples du crâne, Bull. Soc. anat. de Paris, series 3, **10**:247, 1875.

Symmetrical fractures from each side of the foramen magnum through the thin bone of the inferior occipital fossae, then forward to each jugular foramen, formed the rear of a ring fracture described by Petit.[45] It was completed in front by petrobasilar diastasis. It followed a fall so as to alight on the head. Von Bergmann [46] described and illustrated an asymmetrical ring fracture with considerable comminution in the back of the base of the skull. He observed it when he was at Dorpat, where so many studies of cranial fractures were subsequently made (Schwartz, 1872; Hermann, 1881; von Wahl, 1883; Greiffenhagen, 1887, von Knorre, 1890, and others). It was produced by a fall from a great height. He found accounts of only 5 other ring fractures at that time. Laurand [47] found complete detachment of each occipital condyle by fractures surrounding them closely. A branch ran out to the groove for the lateral sinus from the left small pericondylar ring, and from the right ring, a branch ran to the mastoid. The right petrobasilar suture was torn open, and through the middle fossae and body of the sphenoid bone a transverse fracture coursed. These injuries were in the cranium of a coachman who fell head first to the pavement while trying to recover the reins; he died two days later.

Part of an incomplete ring fracture in an illustration by Schranz [48] includes a transverse fracture of the clivus; it was made experimentally by indirect violence to imitate force transmitted by the spine. Hermann [49] found 61 reports of skull fractures in the medical literature published subsequent to the review by Schwartz ten years previously. These fractures, with 8 broken skulls he found in the Dorpat collection, 5 furnished him by Körber and another by Luken, together with the fractures he made experimentally in 17 heads, formed the basis for his study. He divided basilar fractures into transverse, lengthwise, diagonal, ring and nondescript. In his 92 cases, there was no ring fracture. One of the 2 incomplete ring fractures in the illustrations accompanying von Wahl's article [50] was found in Lukin's collection at Kronstadt. The fracture was caused by a fall head first on a stone floor, and was incomplete

45. Petit, C. H.: Fracture de la colonne vertébrale; fracture indirecte de la base du crâne; fracture incomplète de la première pièce du sternum, Bull. Soc. anat. de Paris, series 3, **10**:62, 1875.

46. Von Bergmann,[38] p. 129, fig. 24.

47. Laurand, G.: Fracture du crâne, probablement par contre-coup; rupture de l'artère miningée moyenne, Bull. Soc. anat. de Paris, series 4, **4**:721, 1879.

48. Schranz, J.: Untersuchungen über die Entstehung von Schädelbrüchen, Med. Jahrb., Wien, 1881, p. 291, plate XII.

49. Hermann, N.: Experimentelle und casuistische Studien über Fracturen der Schädelbasis, Inaug. Dissert., Dorpat, 1881.

50. von Wahl, E.: Ueber Fracturen der Schädelbasis, Samml. klin. Vortr., 1883, no. 228 (Chir. no. 73), p. 1945; figs. 18 and 19, p. 1969.

behind. The other fracture, incomplete in front, was in a skull in the collection of the surgical clinic in Dorpat. How this skull was broken is not stated. The fractures in each skull belong to the small ring variety, and from each ring other fractures radiated.

One ring fracture is described and illustrated in the first of the two famous monographs by Messerer.[51] Another he demonstrated during an address at Munich.[52] Both were made experimentally and are reproduced here to illustrate the difference between them and the ring fractures commonly produced by human casualties. The first (fig. 3) was made by sawing off the skull cap and compressing the lower half of the skull with hydrostatic pressure in a Werder machine which

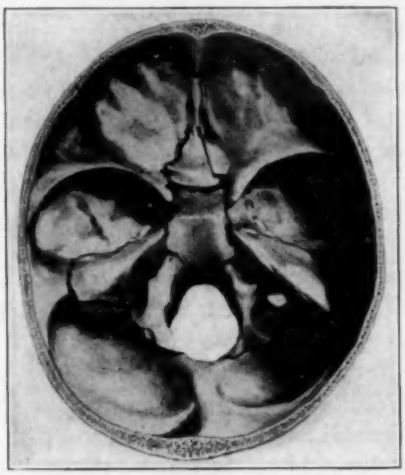

Fig. 3.—A ring fracture produced by Messerer. The calvarium was removed, and the base compressed in a Werder machine. (From Messerer: Ueber Elasticität und Festigkeit der menschlichen Knochen, Stuttgart, 1880, fig. 1, plate VII.)

recorded the force used. Messerer did not state how the second fracture (fig. 4) was produced. It is not illustrated or described in his monographs. A third ring fracture[53] was made by dropping a weight on top of a head still normally attached to the body. From the place hit, a fracture ran squarely across the vault into the right middle fossa, ending in the right carotid foramen. Around the foramen magnum there was a ring fracture enclosing the clivus, the left petrous bone

51. Messerer, O.: (*a*) Ueber Elasticität und Festigkeit der menschlichen Knochen, Stuttgart, J. G. Cotta, 1880; (*b*) Experimentelle Untersuchungen über Schädelbrüche, Munich, M. Rieger, 1884.

52. Messerer, O.: Ueber die gerichtlich-medizinische Bedeutung verschiedener Knochenbrüche-Formen, Friedreich's Bl. f. gerichtl. Med. **36**:81, 1885.

53. Messerer,[51b] p. 12, experiment 66.

and both anonymous processes of the rim of the foramen magnum. Messerer did not state its course on the right side; it probably followed the petrobasilar suture. In his experiment 68,[54] he also broke the skull across with isolated fractures of each anonymous process and a separate fracture lengthwise in the clivus. This, Messerer labeled ring fracture. There are no illustrations of the ring fractures in experiments 66 and 68. In 2 other skulls, independent fractures of the base were produced, which Messerer regarded as suggestions of ring fractures.

Dulles summarized the particulars of the fractures in 119 craniums. In his final illustration,[55] the course of 100 fractures is represented in the customary single diagram of the inside of the base of the cranium. One cannot decide whether in the great number of lines in this composite picture there are included many representing ring fractures about the

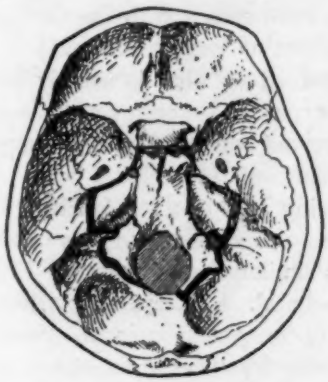

Fig. 4.—A ring fracture Messerer showed at Munich in 1885. Judging from its description, it was produced by a falling weight. (Messerer: Freidrich's Bl. f. gerichtl. med. **63**:81, 1885.)

foramen magnum. In examining the literature, as he must have done to assemble so many fractures, Dulles undoubtedly saw accounts of a number. Perhaps the best indication of the degree of his interest in these ring fractures is in this statement:[56]

> Finally I have been struck by the fact that certain peculiar fractures of the base seem to have been due to support of a segment of the occipital bone furnished by the inclination upward and outward of the articular surfaces of the atlas which embrace the condyles of the occipital bone and restrain it from rupture to such an extent that a fracture will pass on both sides of the protected region rather than through it. This form of fracture I illustrate by a beautiful specimen taken from the Mütter Museum.

54. Messerer,[51b] p. 12.
55. Dulles,[10b] plate XXVII.
56. Dulles,[10b] p. 308.

But to which illustration he referred is not evident.[57] In one figure[58] an incomplete ring fracture is shown about the cribriform plate, and presumably this is alluded to in his statement: "One shows a ring fracture, in addition to . . ." At this time, Dulles' interest was mainly in the experiments by Messerer and others published shortly before. A number of years later, he was more explicit about ring fractures. He said:[59]

> An interesting form of violence, applied to the skull is that due to falls on the feet, where the momentum of the body is suddenly arrested by the resistance of the earth. In such a case a ring of bone surrounding the condyles may be driven into the skull.

Körber,[60] in medicolegal work at Dorpat, found 2 ring fractures, both quite symmetrical, about the foramen magnum. One was inside the other in the head of a man struck with the falling wing of a windmill. The outer ring was about as large as it could be in its posterior half and also remained confined to what is usually included in the bottom half of the cranium. It surrounded each petrous bone and the dorsal part of the body of the sphenoid bone. The inner ring included the occipital condyles and pars basilaris of the occipital bone. Also symmetrical and quite like this inner ring was another fracture Körber found in the body of a boy 10 years old, whose head was crushed against the spine by machinery. It was similar to ring fracture 25 (fig. 6) in our series but much more symmetrical. Körber's ring crossed the middle of the internal median crest in the posterior fossa. From all 3 ring fractures described by Körber, other fractures radiated.

In the article by Anna Heer[61] a small ring fracture is illustrated which began back of each occipital condyle with its front across the clivus from the apex of one petrous bone to the other. Passing out from each side of the ring were symmetrical fractures that broke the petrous bones across in their outer halves and ran forward toward the sphenoid fissures so as almost to complete a second ring in front of the first. From this second unfinished ring, a fracture radiated up in the vault on the right side, parallel with the coronal, and almost to the sagittal,

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57. The course of each of the 119 fractures was shown by drawings he presented to the College of Physicians in Philadelphia. Such separate treatment was not carried out in his published article.

58. Dulles,[10b] plate III and p. 314.

59. Dulles: Remarks on Fractures of the Skull, M. News **68**:81, 1895.

60. Körber, B.: Gerichtsärztliche Studien über Schädelfracturen nach Einwirkung stumpfer Gewalten, Deutsche Ztschr. f. Chir. **29**:544, 1889, plates 10,10A and 10B, 11, 13A and 13B.

61. Heer, A.: Ueber Schädelbasisbrüche, Beitr. z. klin. Chir. **9**:1, 1892, fig. 7, plate II.

suture. A fall of from 9 to 10 meters caused the injuries. The scalp wound was over the right parietal bone close to the vertex.

Chipault and Braquehaye [62] referred to a ring fracture presented by Valude to the anatomic society of Paris and illustrated it with a diagram of the outside of the base of the skull. It began behind each occipital condyle, and on the right side ran along the petrobasilar suture, then obliquely across the front end of the basilar plate to the apex of the left petrous bone; from here it ran through the left carotid canal to the front of the left petrous bone, and across this bone to the left jugular foramen, then behind the left condyle. From its inner side, branches sprang so that each occipital condyle was completely and closely encircled by a small ring within the larger one. Chipault and Braquehaye did not give any reference as to where Valude's observations were published, nor did they tell how the injury occurred.

Van Nes portrayed a partial ring [63] formed by two symmetrical fractures, one on each side of the clivus. They ran from the carotid canals as far back as the outermost part of the foramen magnum, ending in the inferior occipital fossae. They were convex outwardly and would have been parts of a small ring, had it been complete. They were in the skull of a man who jumped through a window, alighting on his feet.

Symmetrical fractures in 4 skulls are described by Phelps, but there are no diagrams or other illustrations. One [64] fracture was due to a fall on the sidewalk. "The whole central portion of the occipital bone from the foramen magnum upward, and posterior portion of both parietal and right temporal bones, forming an irregular circle from two inches and a half to three inches in diameter, were broken into large fragments." Among the 80 fractures in our illustrations, there is none like this. In the other 3 skulls [65] fractures ran on each side of the foramen magnum from the occipital fossae, but failed to form rings. Phelps did not learn how these last 3 fractures were produced. A ring fracture which surrounded all the occipital and right petrous bones with its front end across the sella turcica was produced experimentally by Quenu and Tesson.[66] The dead body was thrown violently backward so that the occiput struck.

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62. Chipault and Braquehaye,[35] p. 292, fig. 19.

63. van Nes: Ueber Schädelbasisbrüche, Deutsche Ztschr. f. Chir. **44**:593, 1897, fig. 2.

64. Phelps,[19b] case 7, p. 397.

65. Phelps,[19b] case 87, p. 446; case 88, p. 447; case 92, p. 449.

66. Quenu, E., and Tesson, R.: Des fractures postéro-antérieures para-médianes de la base du crâne, Rev. de chir., Paris **23**:577, 1901. "Pièce no. 4."

In the English translation of the "Handbuch der praktischen Chirurgie," [67] a small ring fracture is shown, with its front across the clivus and a fracture radiating from its left side through all of the base in front of the ring and then "extending to the frontal bone." Its course in the calvarium is not stated or illustrated. How it happened is not related. The patient died in von Bergmann's clinic.

Bohl's [68] references to ring fractures are chiefly confined to repeating statements by Körber about bending fractures of the base, that they are complete or incomplete rings made by compressing the sides, the front or back of the skull against the spine. Barnick's theme [69] was traumatic intralabyrinthine hemorrhage with basilar fractures. One of his 4 cases dealt with bilateral comminution in the posterior fossa with fractures leading out from each side of the rim of the foramen magnum, suggestions of rings and a somewhat symmetrical arrangement. They resulted from a fall of 3 meters. The superficial injuries were in the back of the scalp and of the right elbow. Stierlin [70] found at a postmortem examination what he regarded as calluses on each side of the foramen magnum and attributed them to a fall from a load of hay seven and a half weeks previously; a clinical diagnosis of ring fracture had been made, presumably from symptoms of injury to the ninth and twelfth cranial nerves.[71] There were other fractures in the base sagittally directed.

Graf's review [72] of 90 basal fractures is mainly clinical. There were 28 deaths and only 11 necropsies. Only 1 ring fracture is mentioned. Its course is described as close to nearly all the rim of the

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67. von Bergmann, Ernst; von Bruns, P., and von Mikulicz, J.: A System of Practical Surgery, translated and edited by William T. Bull and Walton Martin, Philadelphia, Lea Bros. & Co., 1904, p. 77, fig. 18.

68. Bohl, E.: In Sachen der Schädelbrüche, Deutsche Ztschr. f. klin. Chir. **43**:537, 1896.

69. Barnick: Brüche des Schädelgrundes, Arch. f. Ohrenh. **42**:22, 1897.

70. Stierlin, R.: Schädelbasisfraktur mit Lähmungen im Gebiete des X und XII Hirnnerven, Arch. f. klin. Chir. **61**:130, 1900.

71. In this connection see: Deroubaix.[16] Spiller, W. G.: A Physiological, Anatomical and Pathological Study of the Glossopharyngeal and Vagus Nerves in a Case of Fracture of the Base of the Skull, M. Bull. Univ. Pennsylvania **16**:13, 1903. Siebenmann, F.: Eine gleichzeitige Lähmung der Vagus-Accessorius-Glossopharyngeusgruppe als Folge von Schädelbruch, von Erhängungsversuch und von Sinusthrombose, Ztschr. f. Ohrenh. **65**:114, 1912. Lüken, E. A.: Ein-und gleichzeitige Vagus und Accessoriusläsion und vollkommene Taubheit nach Schädelbasisfraktur, Arch. f. klin. Chir. **104**:1000, 1914. Ulrich, K.: (*a*) Ueber Vagus, -Facialis, -und Acusticus-Verletzungen: Ein Beitrag zur Lehre von den Gehirnnerven-Läsionen bei Schädelbasisbrüchen, Schweiz. med. Wchnschr. **3**:545, 1922; (*b*) Verletzungen des Gehörorgans bei Schädelbasisfrakturen, Acta oto-laryng., 1926, supp. 6.

72. Graf: Deutsche Ztschr. f. Chir. **68**:464, 1903, case 9.

foramen magnum, except where it ran out in the tabular part of the occipital bone. It was caused by a fall of 2 meters so as to alight on the head, and there was a small scalp wound above the occipital boss. Brun has 1 ring fracture in his long account of 470 skull fractures seen in the surgical clinic at Zurich from 1881 to 1901. There were 165 deaths. The ring fracture that he described and illustrated [73] was fairly large, through the middle of each petrous bone, the body of the sphenoid bone in front and close to the foramen magnum behind, being almost symmetrical with a fissure behind from the ring to the vault. It was due to a fall from a roof. The external injuries are not mentioned.

Among the diagrams in the article by Poulain,[74] 2 fractures are outlined which he included among transverse fractures. The first is credited to Jacob; the second to Borden, both without reference. These fractures cross the midline in or near the sella turcica and course so far along or within the front of the petrous bones that they may be regarded as the ventral halves of large ring fractures. A third fracture, also from Borden, was composed of three fourths of a large ring, incomplete from the right jugular foramen to the sella. There is no account by Poulain as to how these fractures were sustained.

The ring fracture encountered by Sharpe [75] was asymmetrical and accompanied by fractures of the pars basilaris, and by others radiating up in the vault. The injury was due to an aeroplane accident. A tub or vat falling on the head produced the large ring fracture illustrated by Reuter.[76] His figure is one of the relatively few representing the course of a fracture in the outside of the base of the skull. Vance [1] recognized two forms of skull fractures: linear and composite. Among those in the vault are also depressed fractures. He also grouped them as to location. He mentioned ring fractures in the following quotation:

> Two freak examples of fracture occurred which were included, for the sake of convenience, among the composite fractures of the vault and base. The force which produced them, however, was unusual. The persons in question fell, striking on the vertex of the head, and both sustained fractures of the base, around and just anterior to the foramen magnum. There was evidence that the weight of the body, transmitted through the spinal column, forced the articular process of the occiput up through the base of the skull for a short distance.

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73. Brun,[2b] case 156, fig. 144, p. 328.

74. Poulain, J.: Mécanisme et classification des fractures de la base irradiées à plusieurs étages, Arch. gén. de chir. **8**:884, 1912; first fracture, fig. 12, p. 898; second fracture, fig. 21, p. 899, and third fracture, fig. 38, p. 911.

75. Sharpe, N. W.: Piston Action of Vertebral Column Developed in Certain Types of Cranial Fracture, Bull. Johns Hopkins Hosp. **29**:281, 1918.

76. Reuter, K.: Ueber die Folgen der Einwirkung Stumpfer Gewalt, in Lochte: Gerichtsärztliche und polizeiärztliche Technik, Wiesbaden, J. F. Bergmann, 1914, fig. 118, p. 444.

Vance has no illustrations of these fractures. Among his diagrams of types of fractures is an incomplete and a complete ring.[77]

The foregoing brief from the literature on ring fractures embraces 45 more or less complete ring fractures; 23 were caused by headlong falls of some distance so as to strike head first; 1, that reported by Phelps, by a fall on the sidewalk; 1, by a fall so as to alight on the feet; 4, by blows on the head and 1, by head-crushing; 8 were produced experimentally and there is no information as to the mode of production in 7. The noteworthy feature is that 23 were caused by falls from several feet or more. The review is probably not exhaustive, but it indicates the relative frequency of observations of ring fractures up to modern times, the consideration they have received and the way they have been explained. In our series of 80 fractures there are 5 ring fractures of the vault and 36 of the base. Some of the latter are large; others are small.

REVIEW OF THE FRACTURES IN OUR SERIES

Large Ring Fractures of the Base of the Skull (20).—This type of ring fracture splits from the skull a large concave disk in the base, mainly of occipital bone, with the center of the disk near or opposite the top of the spine (*1* to *15,* and *16* to *20*).[78] The ring is often finished behind by traumatic diastasis of the lambdoid suture, in front by fissures coursing in the petrous bones lengthwise, or along their ventral borders, and across the body of the sphenoid bone or transversely through the firm union between this bone and the basilar occipital plate. Externally, the rings run close to the base of the petrous bones, and a number are continued forward by diastasis of the parietotemporal sutures. Exceptions to this ground plan occur, more of them with the lateral than with other parts of the ring. The ring is sometimes narrowed so that the fracture is obliquely or more directly across one or both petrous bones, but its front and back locations remain fairly constant. Occasionally, removal of the front slightly to the rear brings this part across the basilar plate (*4* and *10*), but this is rare as compared with the frequency of invasion of the petrous bones. In only 1 of this group of 20 skulls was the fracture so low behind that the ring was all in the base, and in this cranium comminution split the ring and the other parts of the fracture were in the calvarium (13).

On the other hand, lateral deviations from the remarkable symmetry of 1 fracture (*1*) were fairly common. They varied widely from a narrowing which left the middle fossae intact (*3*) to asymmetrical

77. Vance,[1] fig. 3 D and E, p. 1028.

78. From here on numbers in the text used as these are indicate the fractures in figures 5 to 9 and the cases in tables 1 to 5, inclusive.

fractures of the petrous bones (*3, 4, 10, 12* and *18*) and to large ring fractures with some comminution of the base (*14, 15* and *18*). A few of the large ring fractures were not quite complete behind (*8, 9, 10* and *11*); others (*3, 16* and *17*) were incomplete in front. In the base of 1 skull (*17*) a huge ring fracture enveloped the middle fossa ventrally. Behind where it joined the back ring, both were incomplete on this inner surface. A word of caution is necessary at this point, because relatively few examinations were made of the *outside of the base* of the cranium,[79] and some ring fractures that were imperfect in the internal table owing to a small gap may have been completed in the external layers of the base. Many descriptions of fractures in the literature leave no doubt that the entire skull was available for study. Accompanying the accounts by some authors there are illustrations from which one may infer that the skulls were museum preparations. On the other hand, it is true that the conditions in the outside of the base are seldom mentioned. In the calvarium, absence of symmetry comparable with that in the base was occasionally encountered, and sometimes the top piece was comminuted (*5, 6, 7, 13, 14, 15* and *20*). In the base extensions ran forward from a few ring fractures (*12, 14, 15 17, 18* and *20*), and a few such ventral extensions were sagittal in either the base (*14*) or the vertex (*19*). In 3 of the 20 skulls, there were independent fractures of the orbital roofs (*2, 4* and *9*). More or less loosening of an entire petrous bone is fairly common with basilar fractures; in fact, it is more frequent than is intimated by the particulars concerning the 20 large ring fractures. Breaking away of the posterior clinoid processes from the rest of the sphenoid bone (*1, 2, 3, 8, 13* and *20*) has been a subject of frequent comment by many observers. It is said that they are pulled away by the tug of the tough tentorium cerebelli when the sagittal axis of the base is lengthened by violence. Another feature of interest, also usually accredited to the careful observations of Félizet,[15] is the firm union between the apex of the petrous bone and the portion of the basilar plate of the occipital bone to which it is applied and the strong connection with the body of the sphenoid formed by the fibrocartilage of the carotid canal. These symphyses usually prevent traumatic diastasis of the petrous apexes, and fractures in that region commonly cross so as to break off the inner third of the petrous bone. This portion of each bone with the basilar plate and a zone about the foramen magnum including the occipital

79. If the back flap of the scalp is dissected from the skull all the way down to the spine, and the skull disarticulated from the atlas, it may be dislocated and lifted so that the base is perpendicular and faces dorsally. The course of fractures in the inferior surface is then easily seen. The skull is replaced on, and wired tight to, the top of the spine, and no external sign remains to indicate the thoroughness of the examination.

TABLE 1.—*Data on Fractures in Figure 5*

No., Sex,* Age	Weight, Height	External Injuries	Injuries of the Brain; Length of Fracture, etc.	Circumstances, Observations, etc.
1 ♂ 30 yrs.	112 lbs. 5 ft. 4½ in.	Laceration of the scalp, 24 mm. long, its center 5 cm. cephalad to the inion; bruised buttocks	Tears of the inferior surface of the frontal lobes, the front tip of the right frontal lobe, inferior surface of the right temporal lobe and base of the left temporal lobe; length of fracture, 54.5 cm.	A fall down stairs; bleeding from the left ear; bloody spinal fluid; coma; right-sided facial paralysis; in the hospital 77 hours, 5 minutes; diagnosis, skull fracture
2 ♂ 40 yrs.	112 lbs. 5 ft. 1 in.	Fresh bruise, 15 mm. in diameter, 5 cm. above and 2 cm. to the right of the inion; bruised right hand, and bruised tissues overlying the coccyx and right scapula	Bruised tips of the frontal lobes; torn left temporal lobe and bruised outside of the left cerebrum; traumatic hemorrhages in the pons; the left tympanum torn; blood in the left middle ear; independent fractures of the orbital roofs; length of main fracture, 73 cm.	A fall downstairs striking the cement floor; walked back up stairs; bleeding from one ear; death en route a few hours later
3 ♂ 9 yrs.	47 lbs. 3 ft. 11 in.	Bruises behind each ear, above and below the left eye and of the right hand and leg; fractured right tibia, fibula and clavicle	No noteworthy gross injury of the brain; length of fractures, 42 cm.	Struck by a street car and dragged some distance; fractured right leg; bleeding from the nose and ears; in the hospital 77 hours, 5 minutes; diagnosis, skull fracture
4 ♂ 52 yrs.	142 lbs. 6 feet	Abrasions above each eyebrow, of the right cheek and of the left angle of the mouth; fractured right 8th rib	Torn frontal poles of the cerebrum, left temporal lobe; contusion of the right temporal lobe, and of each half of the cerebellum; length of fractures, 33 cm.	Struck by a street car; blood in the nose; coma; lessened corneal reflexes and strength in the right extremities; in the hospital 6 hours, 10 minutes; diagnosis, basal skull fracture
5 ♂ 70 yrs.	147 lbs. 5 ft. 8½ in.	No fresh external injury anywhere of the outside of the body	Contusion of the anterior pole of each frontal lobe; torn superior longitudinal sinus of the dura; length of fractures, 49.5 cm.	A fall in a livery stable; blood and cerebrospinal fluid from the ears; stupor; vomiting; involuntary defecation; in the hospital 7 days; diagnosis, skull fracture
6 ♂ 45 yrs.	160½ lbs. 5 ft. 8 in.	Vertical abrasion of the nose, 17 by 3 mm.; hemorrhage in the deep scalp tissues behind; no external injury of the scalp	Top of the brain red from trauma; lacerated left frontal lobe tip; smaller lacerations of the right and left temporal lobes; length of fracture, 62.5 cm.	Found unconscious on the street bleeding from the ears and nose; pupillary reflexes absent; coma; bloody spinal fluid under pressure; in the hospital 8 hours; diagnosis, skull fracture
7 ♀ 42 yrs.	137 lbs. 5 ft. ½ in.	Blood in the deep scalp tissues behind; bruised left knee, right hand and arm; no external injury of the scalp	Small tears in the tips of each occipital lobe behind; subarachnoid hemorrhage anterior to the fissures of Rolando in the midline of the top; fluid blood in both middle ear cavities; length of fractures, 73.5 cm.	Hit by a touring car and removed unconscious from the street; death en route to the hospital

8 ♂ 63 yrs.	140 lbs. 5 ft. 2 in.	Bruise behind the right ear; abrasion near the outer canthus of the left eye; abrasion of the right leg; fractured left femur; six right and two left ribs fractured	Bruised external surface of the right side of the cerebrum and lacerated left frontal lobe; length of fracture, 38 cm.	Struck by a motor truck; in shock on entrance with vomiting and bloody cerebrospinal fluid from the left ear; in the hospital 73 hours, 35 minutes; diagnosis, skull fracture and fractured left femur
9 ♂ 50 yrs.	71½ lbs. 5 feet	Superficial abrasions of the left leg and right buttock; no external injury of the scalp	Bruised tips of the left temporal and of each frontal lobe; blood in the mastoid air cells; empty middle ear cavities; independent fractures of the orbital roofs; length of main fracture, 42 cm.	An assault; incoherent on entrance; bleeding from the ears; normal pupils; bloody spinal fluid; in the hospital 12½ days; diagnosis, skull fracture and alcoholism
10 ♂ 67 yrs.	103½ lbs. 5 ft. 6½ in.	Bruised right arm and elbow, and the right side of the face	Bruises with hemorrhage in both frontal and both temporal lobes; length of fracture, 72 cm.	A fall on the sidewalk; coma; bleeding from the left ear; bloody spinal fluid; rigid neck; involuntary urination; in the hospital 3½ days; diagnosis, skull fracture
11 ♂ 54 yrs.	190 lbs. 5 ft. 7 in.	Scalp torn 3 cm. to the right and 4 cm. above the inion; lacerations of the right wrist, the back of each hand and elbow, and above the right eye	Torn tips of both frontal and both temporal lobes; length of fracture, 49.5 cm.	A fall "down some steps"; coma; bleeding from the nose, mouth and left ear; very bloody spinal fluid; all reflexes absent in the extremities; in the hospital 25 hours, 35 minutes; diagnosis, skull fracture
12 ♂ 31 yrs.	169½ lbs. 5 ft. 8½ in.	Abrasion 8 mm. in diameter, its center 10 cm. cephalad and 9 cm. dorsal to the left external auditory meatus; torn right lung; slight bilateral hemothorax	Bottom and sides of the right frontal and temporal lobes bruised and soft; length of fracture, 59 cm.	Hit by an automobile; coma; bleeding from the nose, mouth and right ear; flaccid extremities; lessened reflexes; bloody spinal fluid; in the hospital 15 hours, 55 minutes; diagnosis, basal skull fracture
13 ♂ 17 yrs.	114½ lbs. 5 ft. 2½ in.	Abrasion of the scalp, 2 by 4 cm., its center 2 cm. cephalad and 1 cm. to the right of the inion; abrasion 2 cm. to the left of the vertex, 15 by 5 mm.; bruises about the left eye, of the left shoulder and of the right elbow	Bruised outside of the left occipital lobe; bruised front tip of the right cerebral hemisphere; length of fracture, 71.5 cm.	Fell from an automobile; coma; unequal pupils; blood in the right ear, nose and pharynx; bloody spinal fluid; in the hospital only 55 minutes; diagnosis, skull fracture, right middle fossa
14 ♀ 35 yrs.	115 lbs. 4 ft. 11 in.	Bruises 6 cm. above the opening of the left ear; bruised lower lip, left side of face and forehead, lower extremities, and left shoulder; fractured mandible, right tibia and fibula; fractured 5th cervical vertebra and fifteen ribs	Blood in the leptomeninges of the left cerebral hemisphere and bottom of the right temporal lobe; brain not bruised outside; 547 Gm. of blood in the left pleural cavity; blood in the middle ear cavities, sphenoid and ethmoid sinuses	Struck by a taxicab; pronounced dead in the receiving room of the hospital
15 68 yrs.	133 lbs. 5 ft. 8½ in.	No external marks of violence; five left and seven right ribs broken	Bruised tip of the right frontal lobe, the inferior surfaces of the left frontal and temporal lobes, and the right hemisphere of the cerebellum; length of fractures, 76.2 cm.	Hit by an automobile; persistent bleeding from the left ear; pupils dilated; eyes fixed; muscular twitching; bloody spinal fluid; Cheyne-Stokes respiration; in the hospital 6 hours; diagnosis, basal skull fracture

* In this and the following tables, ♂ indicates male and ♀, female. Length of fracture means total length of all the fractures.

condyles constitute the "centre de résistance" of Félizet. This is illustrated by 5 fractures (*4, 10, 11, 17* and *18*) which broke the petrous bones across near the apex on one or both sides. Diastasis between these bones and the basilar plate at the front end of one ring (*3, 7, 11* and *15*) is more common in smaller ring fractures.

Small Ring Fractures of the Base of the Skull (*16*).—The transition between large and small ring fractures about the foramen magnum is gradual. The ring in fracture *21* (fig. 6) was a little narrower from side to side, and the ring in fracture *3* (fig. 5) was also narrow, but in the remaining 15 (*22* to *31* and *33* to *37*, inclusive) the rings were definitely smaller. In this series of only 36 more or less complete ring fractures symmetry was not as common in the small as in the large ring type. In a larger number, this relation may be reversed. Only 3 (*29, 36* and *37*) of the 16 small ring fractures were fairly symmetrical. Two of these (*29* and *36*) were associated with more or less comminution of the base. The third ring fracture (*37*) was incomplete in front in at least the inner table. Gaps in 3 other small ring fractures (*21, 24* and *31*) also may have been bridged across by fissures in the outer table. A fourth ring fracture, incomplete behind, is shown in fracture *22* (fig. 6), but ventrally in the two front fossae there was a unique symmetrical ring enclosing the lesser wings, body and a large part of the greater wings of the sphenoid bone. Another equally remarkable feature of the injuries of this cranium was the trival involvement of the calvarium, as compared with the fragmentation of the base. Another base with many fragments is shown in fracture *26* (fig. 6), but in this skull long fractures radiated from behind up in the vault. Fractures *23, 27, 28* and *33* also were continued in the vault, but for such short distances (about equal to the extensions in the skull cap in case *22*), that their diagrams are omitted. In the ring fracture depicted by Sharpe,[75] the fissures within the ring forming a V-shaped fracture of the pars basilaris are the counterpart of that shown in fracture *30*.

In another skull (*28*), this part of the occipital bone was broken transversely. The rings are very small in the fractures represented in *34, 35* and *36*. Bruns,[80] and subsequently Senn,[81] emphasized the danger of sudden death, sometimes days after the accident, from wounds of the medulla oblongata or other parts of the brain stem which are caused by fragments loosened from the rim of the foramen magnum (*26, 31* and *36*). These shocking terminations are said to result from such movements as a sudden turning of the head to one side. This admonition by Bruns and Senn was based on accounts by Bell[27] of

80. Bruns,[14] p. 295.
81. Senn, N.: Fractures of the Skull, Am. J. Surg. & Gynec. **14**:43, 1900.

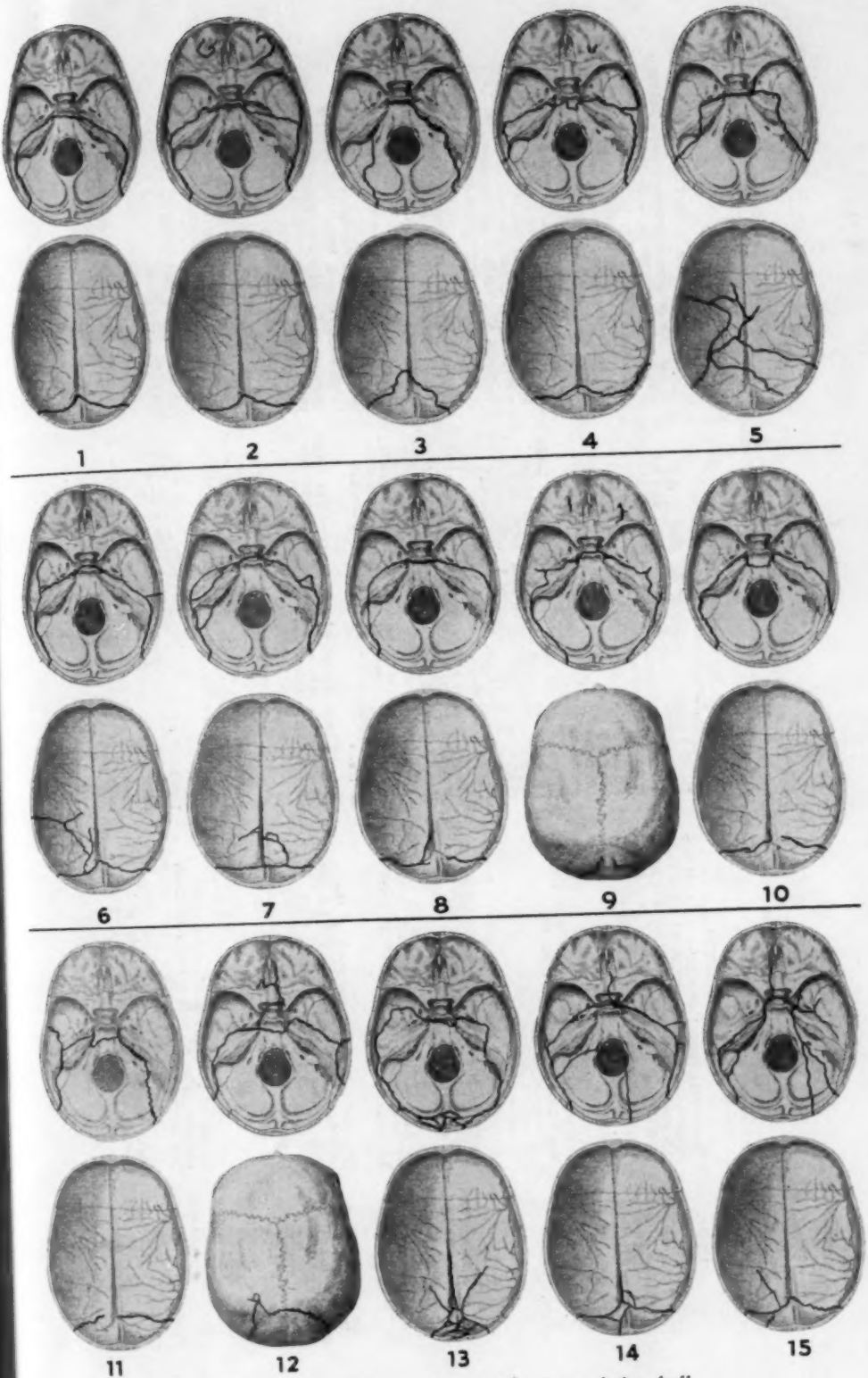

Fig. 5.—Large ring fractures of the base of the skull.

TABLE 2.—*Data on Fractures in Figure 6*

No., Sex, Age	Weight, Height	External Injuries	Injuries of the Brain; Length of Fracture, etc.	Circumstances, Observations, etc.
16 ♂ 32 yrs.	123 lbs. 5 ft. 8 in.	Horizontal laceration of the scalp, 28 mm. long, 4 cm. above the inion, its right end in the midline; bruised back of the left hand; small bruise of the back	Bruised inferior surface of the frontal and temporal lobes; blood in the lateral and 4th ventricles; blood in the right middle ear and right ethmoid cells; length of fracture, 54.5 cm.	A fall downstairs; death soon after entrance to hospital; diagnosis, skull fracture
17 ♂ 27 yrs.	130 lbs. 5 ft. 3 in.	Lacerated wound 6 cm. long above the left eye; bruised left cheek	Bruises of the brain, all dorsal; blood in the orbital tissues; subcutaneous emphysema of the front of the neck; length of fracture not recorded	Hit by a street car; decompression operation on the right temporal fossa; in the hospital 38 hours, 50 minutes; diagnosis, skull fracture
18 ♂ 3 yrs.	26½ lbs. 34 inches	Top of the head bruised; bruise in the deep scalp tissues above the right ear	Scattered microscopic hemorrhages in the brain generally; diastasis of the dorsal end of the sagittal suture 7 cm. long (not shown in the chart); on the left side, a part of the horizontal fracture in the outer table only; length of fracture, 71 cm.	Fell two stories striking head first on a cement court; coma; swollen left occipital region; blood in the pharynx; in the hospital 24 hours; diagnosis, skull fracture
19 ♂ 27 yrs.	118½ lbs. Height not recorded	Scalp wound 10.5 cm. above and 1 cm. dorsal to the right ear, two stitches; no other external injury	Bruised frontal lobes, more on the right side; blood in the right mastoid cells, bottom and front tips of frontal and temporal lobes; linear length of fracture, 69 cm.	Hit by a street car; coma; lacerated scalp; bloody spinal fluid; in the hospital 30 hours, 10 minutes; diagnosis, skull fracture
20 ♂ 38 yrs.	132½ lbs. 5 ft. 5½ in.	No external injury of the scalp; bruised left arm; fractured left ankle and left humerus	Bottom of the brain bruised, both sides equally; torn esophagus and left lung; left hemothorax; blood in both middle ears; length of fracture, 53 cm.	Hit by an automobile; coma; bleeding from the left ear; fractured left humerus and ankle bones; death 5 minutes after entrance to the hospital; diagnosis, basal skull fracture
21 ♂ 83 yrs.	165 lbs. 5 ft. 4 in.	One bruise 1 cm. to the right of the lambda; a second on the top of the head; both bruises small	Bruised frontal poles of the cerebrum; two small bruises of the left side of the cerebrum; length of fracture not recorded	Circumstances unknown; death in the Hospital of the House of Correction at the end of about 3½ days; irrational on admission; diagnosis, delirium tremens
22 ♂ 16 yrs.	97½ lbs. 5 ft. ½ in.	Extensive bruises of the face, more on the right side; fractured left femur	Medulla oblongata severed from the pons with a small spicule of bone in the defect; brain bruises outside, mainly on the right and dorsal; torn apex of the left lung; left hemothorax (310 Gm.); length of fracture, 96.5 cm.	Hit by a motor truck; death en route to hospital
23 ♀ 48 yrs.	92 lbs. Height not recorded	Bruises of the right hand, right elbow and chin; also of both sides of the face, but more of the right side	Hemorrhage in the leptomeninges; brain not torn; 6th to 10th left, and 3d to 7th right ribs fractures; thorax organs not injured; length of fracture not recorded	Hit by a street car; death en route to hospital
24 ♂ 56 yrs.	5 ft. 10 in. Height not recorded	Right wrist bruised; right tibia and fibula broken	Front poles of the cerebrum bruised; fracture entirely in the base; length not recorded	Circumstances not learned; blood in the right ear; in the hospital 24 hours, 35 minutes; diagnosis not recorded

25 ♀ 8 yrs.	45½ lbs. 46½ in.	Bruise, 13 by 15 mm. in diameter, 3.5 cm. above the inion; scattered bruises of the face, right arm and right buttock	One small bruise of each side of the cerebrum; large bruise of the cerebellum; blood in both middle ears, frontal and ethmoid sinuses; length of fracture, 91.5 cm.	Struck by a taxicab; diagnosis of depressed fracture of the left frontal region made in examining room where death occurred
26 ♂ 38 yrs.	144 lbs. 5 ft. 4 in.	Vertical narrow bruise, 2 cm. long, 6 cm. above and 3.5 cm. in front of the left ear; bruised left cheek and left elbow; fractured right leg bones	Left frontal pole of the cerebrum and the bottom of the left half of the cerebellum bruised; length of fractures not recorded	Hit by an automobile; pronounced dead in the receiving room
27 ♂ 30 yrs.	114 lbs. 5 ft. 8 in.	Horizontal tear of the scalp, 5 cm. long, just below the inion, in the midline, and sewed with three stitches; bruised chin and left eyebrow	Bruise of the left frontal lobe 6 cm. deep (front to back); multiple small hemorrhages in the pons; bruised left cerebellar hemisphere; length of fractures, 25.5 cm.	Fell off a street car; coma; restless; bleeding from the right ear; contracted pupils with slow reactions; right nystagmus; bloody spinal fluid; in the hospital 6 hours; diagnosis, skull fracture
28 ♀ 44 yrs.	89 lbs. 5 ft. 2½ in.	Laceration 15 by 35 mm. at the level of the inion and a little to the left; bruise of the left side of the forehead; small bruises of the left knee, left shoulder and the right leg	Both frontal lobes bruised, the right the more; bruises through the gray cortex into the white substance; length of the main fractures, 40 cm.; small independent fracture	Hit by a street car; coma; dilated inactive pupils; involuntary stools; rigid neck; flaccid muscles; bloody spinal fluid; in the hospital 6 days; diagnosis, alcoholism and brain concussion
29 ♀ 19 yrs.	No records	Wound of the back of the head in the midline at the level of the lambda, slightly more on the right	Body decomposed; base of the skull in two lateral halves with the spine projecting into the skull; length of fractures, 87 cm.	Hit on the head with an ax (homicide); found dead
30 ♀ 53 yrs.	113½ lbs. 5 ft. 2½ in.	Scalp torn for 2 by 4 cm., the long axis horizontal, all on the left side, at the inner end at the inion	Under surface of each frontal lobe bruised, the bruises small; small contusions of the bottom of each temporal lobe; blood in the right lateral ventricle; length of fracture not recorded	Fell downstairs; unconscious and cyanotic; bleeding from the mouth, nose and right ear; right pupil dilated; death in the hospital 2 hours after the accident; diagnosis not recorded
31 ♂ Age not recorded	89 lbs. 5 ft. 8½ in.	Horizontal laceration 4.5 cm. long, 1 cm. above and 4 cm. to the left of the inion; a vertical laceration 21 mm. long in the skin covering the left supra-orbital ridge and close to the midline	Small bruise in each side of the cerebrum in the convolutions about the sylvian fissure (truncus fissurae lateralis); hemorrhage in the leptomeninges of the top of the brain; length of fracture, 85 cm.	Circumstances not learned; found dead in the Chicago river
32 ♂ 39 yrs.	105 lbs. 5 ft. 5½ in.	Bruise of the nose and one of the middle of the forehead; fractured third cervical vertebra	Extensive hemorrhage in the spinal leptomeninges caudad to fractured vertebra; bruised spinal cord	A fall in the hospital while under treatment for acute alcohol poisoning; death 3 hours after the fall; diagnosis, acute toxic myocarditis
33 ♂ 49 yrs.	165 lbs. 5 ft. 6 in.	Triangular wound from 18 to 20 mm. on each side in the scalp, 1 cm. to the left of and 6.5 cm. above the inion	Left cerebellar hemisphere torn; small hemorrhages widely scattered in the brain; length of fracture not recorded	Circumstances not learned; no history obtained
34 ♂ 34 yrs.	145½ lbs. 5 ft. 4½ in.	Small purple bruise of the scalp 4.5 cm. to the right of and 4 cm. above the inion; three bruises of the face; blood in left ear	Leptomeningeal hemorrhage; no external bruises of the brain; one small hemorrhage deep in the pons; length of fracture, 46.8 cm.	Found dead in an alley; circumstances not learned
35 ♂ 25 yrs.	123 lbs. 5 ft. 5½ in.	No external injury of the head except an abrasion behind the right ear; blood in the left ear	Small contusions of the inferior part of each temporal lobe; length of fracture, 56 cm.	Death soon after being crushed between the wheel of a wagon and a barn door; not under medical observation

two such deaths. One occurred after it was supposed that the man had fully recovered. He died suddenly "as if he had been pithed . . . . The margin of the great foramen magnum of the occipital bone was fractured, and it appeared that on suddenly turning the head, the condyle was displaced and the loose bone brought to press and nip the medulla oblongata." The other [82] death occurred while the head was being shaved by one attendant, and the patient was being bled by another. The latter noticed that the blood no longer flowed; "the loose portion of bone had been turned upon the spinal marrow, and crushed it." In the head of a man examined by Beck [83] "the bone between the jugular foramen and the margin of the foramen magnum on each side was splintered into several fragments . . . an entirely separate fracture was found in the region corresponding to the condyles of the occipital bone. . . . On pushing the fragments up into the skull the opening of the foramen magnum was considerably narrowed, and this seems to have produced the injury to the floor of the fourth ventricle." The man fell down a staircase that he was painting and was found standing on his head between the wall and the ladder. Shortly before Bruns explained this hazard from the small ring fractures, Trélat [84] also reported a similar unexpected death.

With impartial disposition of fractures on each side of the skull, one might expect a similar balance in the distribution of the occasioning force. So far as the large ring fractures in our series of 80 are concerned (tables 1 and 2, cases 1 to 20, inclusive), the evidence of violence compressing the skull vertically against the spine is not overwhelming.

In only 1 fracture of the group of 20 do the conditions match those commonly described for ring fractures. This was a fracture produced by a headlong fall of some distance to a cement court (*18*). The fracture differs, however, from those usually described, because they are in adults, as a rule, and this fracture was in an infant 3 years old, and also because most of the reported ring fractures caused by similar accidents have been close to the foramen magnum. This infant's scalp was bruised on top. With each of 5 other fractures in the group of 20 (*1, 2, 11, 13* and *16*), the scalp injury was close to the inion, and there were contrecoup contusions of the brain at the front end of the cerebral hemi-

82. Bell, C.: The Nervous System of the Human Body, ed. 3, London, Longmans, 1844, observation 149, p. 403. Both observations by Bell are mentioned by Chassaignac: Lésion traumatique du crâne et des parties qu'il contient, Thèse du concours, Paris, 1842, p. 27.

83. Beck, M.: Fracture of the Base of the Skull, Tr. Path. Soc. London **24**: 181, 1875.

84. Trélat, U.: Fractures du crâne, Bull. Soc. anat. de Paris, series 1, **27**:213, 1852.

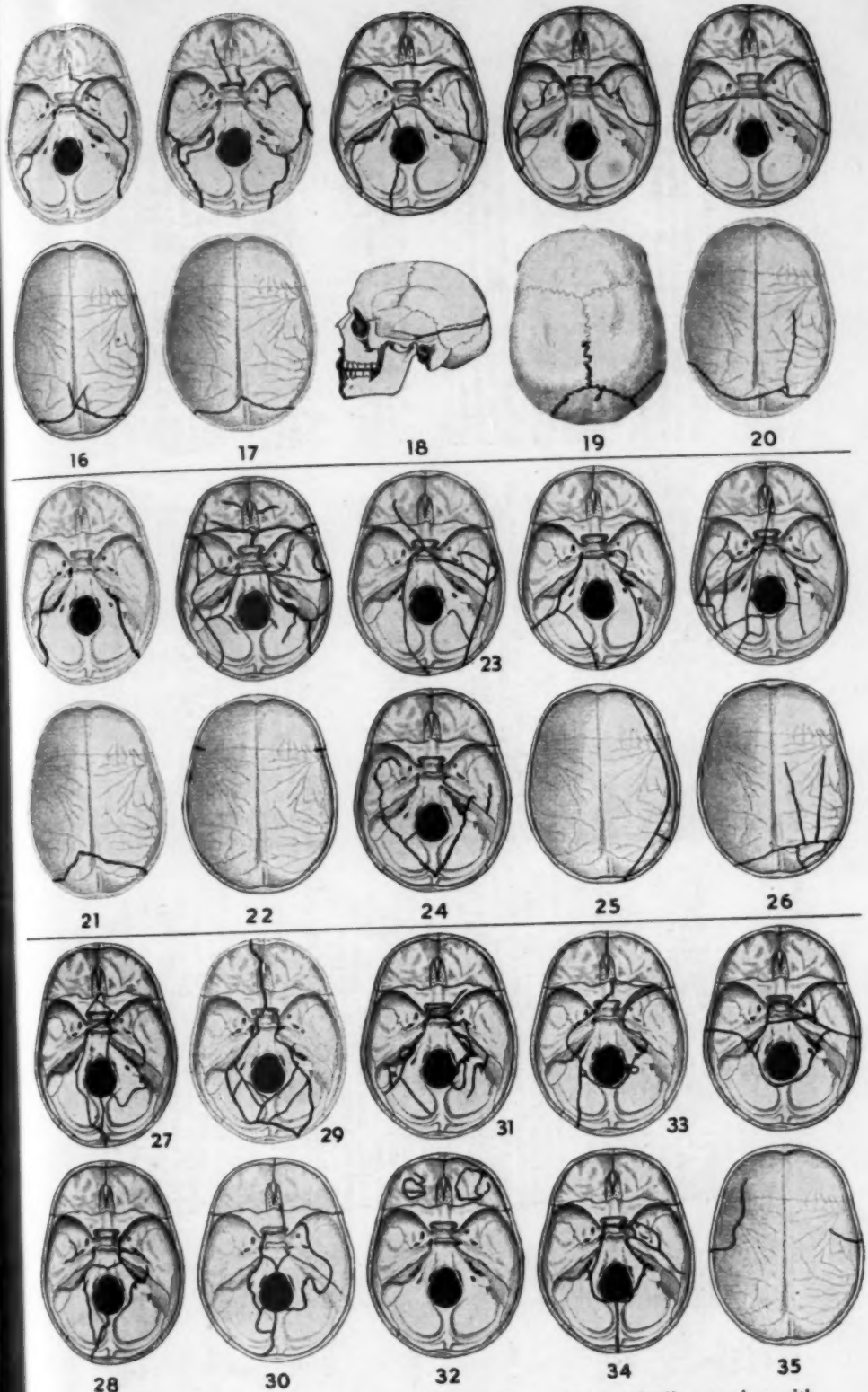

Fig. 6.—Large ring fractures of the base of the skull, gradually merging with smaller ring fractures. Solitary fractures of the orbital roofs are illustrated in *32*. The dotted lines in *18* indicate the course of the fracture on the opposite side.

TABLE 3.—*Data on Fractures in Figure 7*

No., Sex, Age	Weight, Height	External Injuries	Injuries of the Brain; Length of Fracture, etc.	Circumstances, Observations, etc.
36 ♂ 40 yrs.	135 lbs. 5 ft. 7 in.	Bruise 5 cm. in diameter opposite the inion with a laceration in the center of the bruise "down to the bone"; two smaller bruises of the right side of the forehead; bruised right elbow, right knee, left groin and hand	Torn inferior surface of both frontal and of both temporal lobes; also of the vermis of the cerebellum; length of fracture, 89.6 cm.	Hit by an automobile; coma; bloody spinal fluid; in the hospital 33 hours, 20 minutes; diagnosis, anterior basal skull fracture
37 ♂ 30 (?) yrs.	148 lbs. 5 ft. 5½ in.	Lacerated wound 2 cm. in diameter, 1 cm. to the right of the inion; small bruise of the right elbow	Bruised inferior surfaces of both frontal and both temporal lobes, the bruises much larger on the left side; length of fracture not recorded	Circumstances not learned; bleeding from the nose and pharynx; coma; wound of the scalp; in the hospital of the House of Correction 7 hours, 10 minutes; diagnosis, basal skull fracture
38 ♂ 72 yrs.	115 lbs. 5 ft. 7 in.	Laceration of the scalp 3 cm. to the right of the inion with radiating tears (4) from 2 to 5 cm. long; three small lacerations of the left side of the face; purple left eyelids; fracture dislocation of the 5th and 6th cervical vertebrae	Superficial bruises of the inferior surfaces and deep bruises of the medial surfaces of both frontal lobes; a deep bruise in the right occipital lobe; bruised right temporal and parietal lobes (lateral surface); bruised top of the corpus callosum; length of fracture, 38.1 cm.	Hit by an automobile; deep coma; unequal pupils; rigid extremities; bloody spinal fluid; in the hospital 5½ hours; diagnosis, skull fracture
39 ♂ 55 yrs.	132½ lbs. 5 ft. 6½ in.	Horizontal, irregular, deep tear of the left eyebrow 1 inch long; bruises and abrasions of the left side of the face and left side of the scalp ventrally; small contusions of both legs, the left arm, right foot and right hip; three left ribs fractured; fractured right pubic and ischiatic bones	Simple "traumatic lividity" of the brain, i. e., hemorrhage in the leptomeninges; length of fracture not recorded	Hit by a taxicab; coma; small equal pupils; blood in the nares; bloody spinal fluid; in the hospital 2 hours, 45 minutes; diagnosis, skull fracture
40 ♂ 25 yrs.	89 lbs. 5 ft. 3 in.	Eyelids purple from orbital hemorrhage; bruised right knee; 70 Gm. of blood between the dura and the calvarium on the left side, the clot 2 cm. thick	Superficial bruises of the lower frontal and temporal convolutions of each side and of the inferior surface of both frontal and temporal lobes; compressed brain; one fracture 27.5 cm. long; the other, 28.1 cm.	Hit by a taxicab; coma and dyspnea; restlessness; bloody spinal fluid; in the hospital 44 hours, 50 minutes; diagnosis, cerebral concussion, suspected skull fracture
41 ♂ 65 yrs.	116 lbs. 5 ft. 7 in.	Vertical laceration of the front of the right temporal fossa; the scalp abraded 4 cm. to the right and 6 cm. in front of the inion; abrasion of the left cheek; fractured 6th cervical vertebra, pubic bones, left tibia, left fibula and three left ribs	Many small superficial bruises of the inferior surface of both frontal and both temporal lobes; length of fracture, 49.6 cm.	Hit by a taxicab; bleeding from the nose; coma; in a first aid hospital 5 hours, then in Cook County Hospital 4 hours; diagnosis, lacerated scalp and fractured leg bones
42 ♂ 30 yrs.	120 lbs. 5 ft. 4 in.	Right side of the chin bruised; surgical incision of the scalp above and behind the right ear	Small contusion of the under surface of the cerebrum; length of fracture not recorded	Circumstances not learned; comatose; in the hospital 3 hours; diagnosis, suspected skull fracture

43 ♂ Young adult	Wt. not taken 5 ft. 2 in.	Contused left side of the head; left femur fractured at the upper end	Fractures began 2.5 cm. above and 1.2 cm. below the left auditory meatus, and ended in an exactly corresponding place on the right side; length of fractures not recorded	Circumstances not learned; no history or diagnosis obtained
44 ♀ 42 yrs.	106 lbs. 5 ft. 2½ in.	Bruised right elbow, cheek, lip and right side of the nose; severe contusion above the left ear; decompression wound of the right side of the head	Entire under surface of the right temporal lobe torn; small tear of the opposite temporal lobe; extensive hemorrhage into the leptomeninges; bruised central lobule of the cerebellum; length of fracture not recorded	Injured by an automobile; coma; bleeding from the left ear; frequent vomiting; right decompression operation; in the hospital 92 hours; diagnosis, skull fracture
45 ♂ 45 (about)	200 lbs. 6 feet	Blood in both ears, the nose and mouth; bruised right elbow; fracture coursed through external auditory canal	Superficial bruises of the under surface of both temporal lobes, the front pole of each frontal lobe and the back of the right occipital lobe; length of fracture not recorded	A fall down a stairway was the verdict of a coroner's jury; found dead
46 ♂ 40 yrs.	145 lbs. 5 ft. 4 in.	Two lacerated wounds of the right side of the head, one 13, the other 10, cm. long; a third 7.5 cm. long above and behind the left ear; three small lacerations of the back of the right hand; bruises of the left cheek; fractured sternum	Scattered regions of hemorrhage in the leptomeninges; no gross contusions of the brain; blood in both middle ears; length of fracture, 63.7 cm.	Struck by an automobile; coma; bleeding from the nose, ears and mouth; in the hospital 5 hours, 45 minutes; diagnosis, skull fracture
47 ♂ 30 yrs.	202 lbs. 6 ft. ½ in.	Lacerated wound 2.6 cm. long, 7.5 cm. above the opening into the right ear; bruises of outer end of the right eyebrow, right side of the forehead and right leg; eleven fractures in seven right ribs; both innominate bones broken in front, more on the right	Hemorrhage in the leptomeninges covering the back end of each occipital lobe, the upper surface of the left occipital lobe and the bottom of the cerebellum on each side; length of fracture, 31.5 cm.	An automobile accident; deep coma; closed eyes; bleeding from the nose and both ears; pupils inactive; in the hospital 3 hours, 45 minutes; diagnosis, basal skull fracture and shock
48 ♂ 40 yrs.	91 lbs. 5 ft. 6½ in.	Two large abrasions of the left side of the face and one of the left ear; bruised back of each hand and right foot	Two large and two small bruises of the outside of the right temporal lobe; bruised right frontal lobe, medial surface and gyrus cinguli; extensive leptomeningeal hemorrhage; length of fracture, 35.5 cm.	Thrown from an automobile when a tire exploded; sluggish dilated pupils; stertorous breathing; bleeding from nose and both ears; bloody spinal fluid; in the hospital 5 days; diagnosis, basal skull fracture
49 ♂ 45 yrs.	137 lbs. 5 ft. 8½ in.	Two lacerated wounds above and behind the left ear, the longer 3 cm. long; a small bruise of the left side of the forehead, another of the right elbow and a third of the left knee	Small superficial bruises of the right frontal and right temporal lobes; length of fracture, 24 cm.	Circumstances not learned; comatose; bloody cerebrospinal fluid running from the left ear; blood in the mouth; dilated pupils; in the hospital 2½ hours; diagnosis, basal skull fracture in the middle fossae
50 ♂ 73 yrs.	123½ lbs. 5 ft. 5½ in.	Small laceration (1 cm. long) in the scalp in the front part of the left temple; large bruises of the back of each elbow	Deep bruises of the right parietal lobe; a small bruise of the back part of the right inferior temporal convolution; length of fracture, 37 cm.	Struck by a street car; irrational; no bleeding anywhere except from the scalp wound; in the hospital 68 hours, 35 minutes; diagnosis, scalp wound and senility
51 ♂ 21 yrs.	117 lbs. 5 ft. 3 in.	Bruises and abrasions of each side of the head; three small perforating wounds of the scalp above and behind the right ear; fluid blood in both middle ears; left tympanum torn	Extensive bruising of the right central convolutions of the parietal region; multiple small hemorrhages throughout the pons; length of fracture, 65 cm.	Automobile collision; coma; bleeding from the nose, mouth and ears; labored breathing; depressed bone felt above the right ear; in the hospital 24½ hours; diagnosis, skull fracture
52 ♂ 36 yrs.	143½ lbs. 5 ft. 8½ in.	No external injuries, but the scalp was purple where bleeding from the skull fracture had occurred; blood in the middle ears, ethmoid and sphenoid sinuses and in the orbital fat; bruised right knee and arm	Scattered superficial contusions of bottom and both sides of the cerebrum, the largest in the right temporal lobe; length of fracture not recorded.	Slugged by burglars with a hammer; coma; paralyzed right arm; sluggish pupils; stiff neck; swelling behind the left ear; bloody spinal fluid; in the hospital 39 hours; diagnosis, skull fracture

spheres, indicating that when violence broke the skull, the head was in motion and was arrested at that moment.[4a] In 6 other bodies (*4, 5, 6, 9, 10* and *15*), there were contrecoup brain bruises at the front tips of the frontal lobes, but no scalp injuries. One man (*9*) was assaulted; another it was said, fell on a sidewalk (*10*). The distance of the fall in another case (*5*) was not learned. Street traffic caused the other 3 accidents (*4, 6* and *15*). The way blows at or near the occipital boss make large ring fractures about the foramen magnum will be discussed when small ring fractures are reviewed. But it is well at this time to emphasize the existence of evidence of violence to the back of the head in both of the last two groups, 11 fractures altogether. In one group, the evidence consisted of scalp injuries and contrecoup brain bruises; in the other, simply brain bruises.

Five of the 20 large ring fractures were less symmetrical than the others (*8, 12, 14, 17* and *19*). The brain bruises were also less symmetrical, which accords very well. In all 5 bodies, the scalp injuries were definitely on one side, an observation in accord with the first two. In order to explain the ring fractures at the base in these 5 craniums, it is necessary to assume that the head was twisted in some manner when hit. The course of events, let us say, for the man 63 years old (*8*), was possibly as follows: His first injury was close to the outer angle of the left eye. By this, he was unbalanced, and his head was turned to the right. Then he fell so that the scalp was bruised behind the right ear. The lagging brain received a contrecoup bump at the anterior end of the left cerebral hemisphere. But the accompanying large ring fracture is almost symmetrical. If it was caused by the oncoming weight of the trunk driven against the base of the skull, it seems plausible that the head at the time was swung around by, and on, rotating cervical vertebrae, so that compression against the spine was from behind the right ear. Applying this notion of a bent head to another of this group of 5 fractures (*17*), in which the contrecoup bruises were in the back poles of the brain and the external wound was above the left eye, one may suppose that the head was thrown well back and a little to the right when the man hit something face first, and the blow of the spine on the base of the skull made the fracture.

Two other large ring fractures (*3* and *20*) may have been produced entirely by violence to the base of the skull. There are no positive indications of this; in fact, both fractures resulted from vehicular accidents. It rather staggers belief that blows from vehicles may so project bodies that the only force applied to the skull is by way of the spine. On the other hand, bones of the lower extremities were broken in both bodies. The boy's head (*3*) was bruised on each side, but the

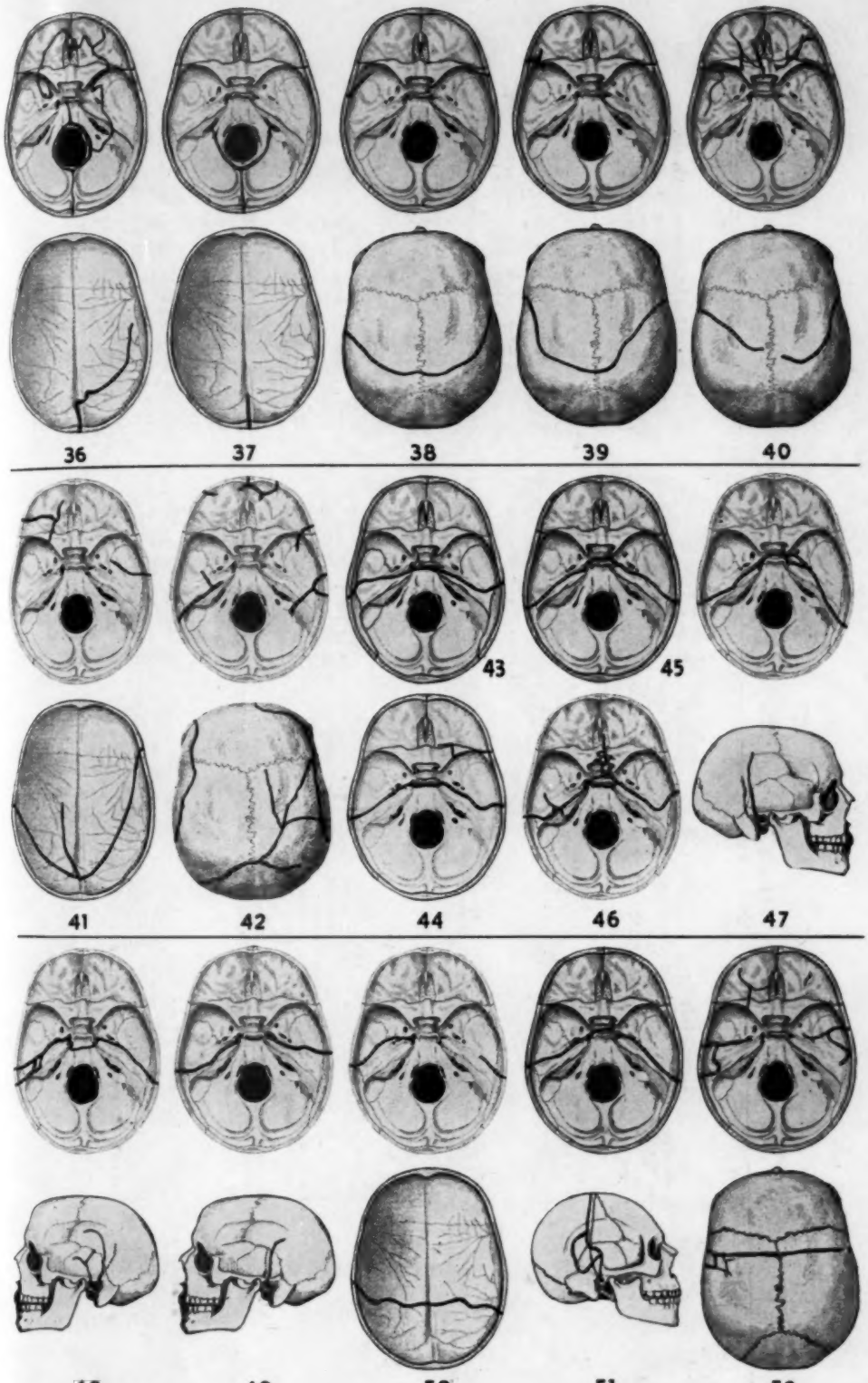

Fig. 7.—Small ring fractures of the base (*36* and *37*), ventral ring fractures of the vertex (*38* to *42*) and dorsal transverse incomplete and complete fractures (*43* to *52*). The dotted lines in *47, 48, 49* and *51* indicate the location of the fractures on the opposite side.

TABLE 4.—*Data on Fractures in Figure 8*

No., Sex, Age	Weight, Height	External Injuries	Injuries of the Brain; Length of Fracture, etc.	Circumstances, Observations, etc.
53 ♂ 41 yrs.	95 lbs. 5 ft. 10 in.	Lacerated wound 15 mm. long above the outer end of the left eyebrow; both temples purple from deep bleeding; bruised upper left arm; sacral bed sore	Extensive but superficial bruises of the right middle and inferior temporal convolutions; torn dura; extradural clot (17 Gm.) compressing the right frontal lobe; acute fibrinous meningitis; length of fracture, 45.9 cm.	Hit by an automobile; mentally confused; spinal fluid clear; time in first hospital not learned; in Cook County Hospital 11 days; diagnosis, skull fracture
54 ♂ 58 yrs.	178 lbs. 5 ft. 9 in.	Lacerated wound 6.5 cm. long up and back from the outer end of the right eyebrow (7 stitches); bruised right side of the face; bruised back of each hand	Hemorrhage in the leptomeninges of the top and side of the left cerebral hemisphere; small bruise of the right frontal and large deep bruises of the left parietal lobes; blood in the lateral ventricles; length of fracture, 17 cm.	Run over by a horse and wagon; coma; left outer strabismus; bleeding from the nose; tongue deviated to the right; blood in the throat; lower extremities rigid; vomiting; bloody spinal fluid; in the hospital 30 hours, 20 minutes; diagnosis, basal skull fracture
55 ♀ 40 yrs.	102 lbs. 4 ft. 10½ in.	Huge abrasion of the right side of the face; fractured mandible 4 cm. to the left of the midline; "black and blue" eyes; bruised left thigh	Minute bruise of the right upper temporal convolution; a larger bruise of the gyrus rectus of the right frontal lobe close to the olfactory trigon; generalized fibriopurulent leptomeningitis; length of fracture, 42.5 cm.	Run over by an automobile; comatose and restless; bleeding from the nose and ears; absent patellar reflexes; recovered consciousness and complained of headache; in the hospital 7 days; diagnosis, basal skull fracture
56 ♂ 34 yrs.	120 lbs. 5 ft. 5 in.	Deep abrasion of the left temple 4 by 2.5 cm.; bruised ears; lacerated wound 1 cm. long in the upper attachment of the right ear; a small abrasion behind the right ear and another on the right side of the forehead; lacerated left upper arm; bruised knees; six fractures in twelve right ribs; five left ribs fractured; hemothorax	Three small contusions of the side of the right and one of the left cerebral hemisphere; bruised anterior tips of each temporal lobe; minute traumatic hemorrhages in the pons and one hemorrhage 8 by 5 mm. in the right thalamus; length of fracture, 42.8 cm.	Fell from a third story porch; coma; profuse bleeding from the mouth and nose; bloody spinal fluid; in the hospital 65 minutes; diagnosis, fractured skull and ribs
57 ♂ 60 yrs.	94 lbs. 5 ft. 1½ in.	Bruise of the right temple 8 by 12 cm.; bruised right upper arm, shoulder, right leg and right side of the trunk	Large deep contusion of the left temporal lobe; length of fracture, 52 cm.	Fell over a stair railing; stupor at first; irrational later; slight bleeding from the left nostril; dilated sluggish right pupil; lacerated tongue; in the hospital 9 days; diagnosis, skull fracture
58 ♀ 28 yrs.	140 lbs. 5 ft. 2½ in.	Irregular laceration 8.5 cm. long in the scalp on top of the head; opposite the coronal suture and transversely directed (8 stitches), most of it on the right side; incised wound of the calvarium and dura	Large deep wound of the top of the brain opposite that in the calvarium; a piece of bone 18 by 12 mm. in the brain wound, length of fracture, 23.7 cm.	Hit on the head with an ax; semiconscious; brain tissue, blood and hair in the wound of the head; flaccid general paralysis; uncontrolled sphincters; bloody spinal fluid; in the hospital one week; diagnosis, open wound of the head and brain

59 ♂ 40 yrs.	Weight not recorded 5 ft. 3½ in.	Hypodermic scars (morphinism) of the abdomen; no fresh external injuries	No injuries of the brain; hemorrhage in the scalp at the vertex, 9 by 6.5 cm.; dura uninjured; length of traumatic diastasis, only 2.5 cm.	Circumstances not learned; sent to the hospital of the House of Correction with delirium and fever; in the hospital 1 day; diagnosis, acute alcoholism
60 ♂ 36 yrs.	121 lbs. 5 ft. 5 in.	Large purple bruise of the forehead on the right side; bruises of the back of each arm, the back of the left hand and inside of the left leg	Deep bruises of the bottom of the frontal lobes and top of the parietal lobes, the former much the larger; superior longitudinal sinus not torn; an extradural hemorrhage (51 Gm.) opposite the top of the frontal lobes due to torn small emissary veins; length of fracture, including the diastasis, 22 cm.	Fell in the hospital while under treatment for alcoholism; swollen scalp on one side; in the hospital 66 hours, 55 minutes; death 44 hours after the fall; skull fracture suspected
61 ♂ 34 yrs.	120 lbs. 5 ft. 5 in.	Surgical decompression wound of the left side of the head; bruise above the left eye; fractured left humerus; large open wound of the left upper arm; loose fragment of bone in the wound; fractured left clavicle	Extradural symmetrical hemorrhage from the glabella to the lambda widest (5 cm.) at its front end; traumatic leptomeningeal hemorrhage ventrally; at the front end the fracture ran in only the outer table to the left supraorbital ridge; dura not torn; length of fracture not recorded	Collided with an automobile while riding a motorcycle; delirious; fractured humerus; in the hospital 72 hours, 35 minutes; diagnosis, fractured left humerus; skull fracture suspected
62 ♂ 34 yrs.	152 lbs. 5 ft. 4 in.	Horizontal bruise-abrasion, 9.5 by 3.5 cm., across the forehead, almost symmetrical but a little more on the right side; bruised right elbow, forearm and hand; fractured 8th to 11th left rib inclusive	Front poles of the temporal and frontal lobes bruised; bruises of the bottom of both frontal lobes, the bruises all superficial but extensive; dura not torn; length of fracture, 25.7 cm.	Hit by an auto truck; restless; coma; stertorous breathing; fractured ribs; pupils active; knee jerks slow; bloody spinal fluid; in the hospital 4 hours, 10 minutes; diagnosis, suspected skull fracture
63 ♂ 5 yrs.	37½ lbs. 45½ inches	Lacerated wound 4 cm. long close to the inion in the scalp; small bruises of both sides of the forehead; left decompression surgical wound; bruised right leg	Deep bruises of the top of each frontal pole of the cerebrum; small bruises of the bottom of the frontal and semporal lobes; small extradural clots opposite the sagittal suture; sagittal dural sinus intact; length of fracture, 29 cm.	Hit by an automobile; coma from which he later could be aroused; decompression operation shortly before death; in the hospital 72 hours, 40 minutes; diagnosis, skull fracture
64 ♂ 40 yrs.	129½ lbs. 5 ft. 4½ in.	Vertical deep laceration of the scalp 4 cm. long, 2 cm. to the right of the inion; four small lacerated wounds of the right and one of the left side of the face; a bruise of the top of the head on the right side; independent fracture of the right external angular process of the frontal bone, floor of the right orbit and superior maxilla; fractured left humerus; five right and two left ribs fractured	Many small superficial bruises of the top and bottom of the frontal lobes, of the bottom of both temporal lobes ventrally and both uncinate gyri; traumatic hemorrhages in the left putamen; length of fracture, 49 cm.	Hit by an automobile; unconscious with a "dissecting hematoma" under the scalp; in the hospital 2 hours, 35 minutes; diagnosis, basal skull fracture
65 ♂ 30 yrs.	180 lbs. 5 ft. 8 in.	No external injury except purple upper eyelids from deep bleeding; blood in the nostrils, esophagus and large air passages	No gross hemorrhages in the brain anywhere; extensive hemorrhages in the leptomeninges of the top of the head; dura not torn; length of fracture not recorded	A fall downstairs; deep coma; bloody spinal fluid; in the hospital 2½ hours; diagnosis, basal skull fracture
66 ♂ 45 yrs.	144½ lbs. Height not recorded	Three bruises of the back of the head close to the inion; right deep palpebral hemorrhage	Large deep lacerations of the under surface of the frontal and front part of the temporal lobes; one minute hemorrhage in the pons; dura not torn; length of fracture not recorded	Fell off a truck, landing head first on a pavement; able to walk but "dazed"; later unconscious; in the hospital 21 hours, 40 minutes; diagnosis, suspected skull fracture

street car had dragged him some distance. His brain was not bruised outside, a condition common with skull fractures in children. The man (*20*) was 38 years old and was hit by an automobile at a street crossing. Although no bones of the trunk were broken, the esophagus and left lung were torn. According to the necropsy record: "About the junction of the esophagus and diaphragm, there is a blood-stained region including the adjacent lung which is irregularly torn from the entrance of the left bronchus to the diaphragm, so that the branching of the pulmonary vein is seen in the bottom of the tear." The tear in the esophagus was small, in line with the tear in the lung and at the diaphragm. The bruises of this brain were confined to the parts in contact with the base of the skull. Obviously of greater importance are the character of the fractures and the absence in each body of injuries of the scalp in the midline.

Evidence to explain another large fracture ring (*7*) is even less satisfactory. One knee was bruised; the scalp was not injured outside. The little loose blood it contained was only 1 mm. thick,[85] opposite the fracture, with its inner limit at the pericranium and obviously from torn vessels in the bone. The injuries of the brain were behind and not contrecoup.

The 16 small ring fractures (*21* to *31* and *33* to *37*, inclusive) may also be examined in groups. With 11, there were associated scalp injuries about the lambda. This is a larger proportion than the 5 in the group of 20 large ring fractures. Because the numbers are small, this difference may have no significance. Another variation is the fewer contrecoup bruises at the front poles of the cerebrum as compared with the frequency of similar contusions of the brain accompanying the large ring fractures. Study of the tables will also disclose more bruises of the bottom of the brain with the small, than with the large, ring fractures. But these are trivial particulars contrasted with the evidence pointing to the production of 23 of the total number of 36 ring fractures,[86] both large and small, by violence applied to the back of the head and not, as one would expect, to the vertex. This evidence, as has been stated, consists of contusions of the scalp at or close to the inion and injuries of the front pole of the cerebrum. Each supplements the other

85. In order to decide whether blood free in the scalp results from external violence at that place or is from vessels in the bone torn by the fracture, parallel cuts close together should be made from the inner surface of the reflected scalp outward, and almost, but not entirely, through the scalp.

86. Among the large ring fractures, *1, 2, 11, 13* and *16; 4, 5, 6, 9, 10* and *15;* among the small ring fractures, *21, 25, 27, 28 29, 30, 31, 33, 34, 36* and *37* with dorsal scalp injuries and *24* with deeply bruised tips of the frontal pole of the cerebrum but no scalp injuries.

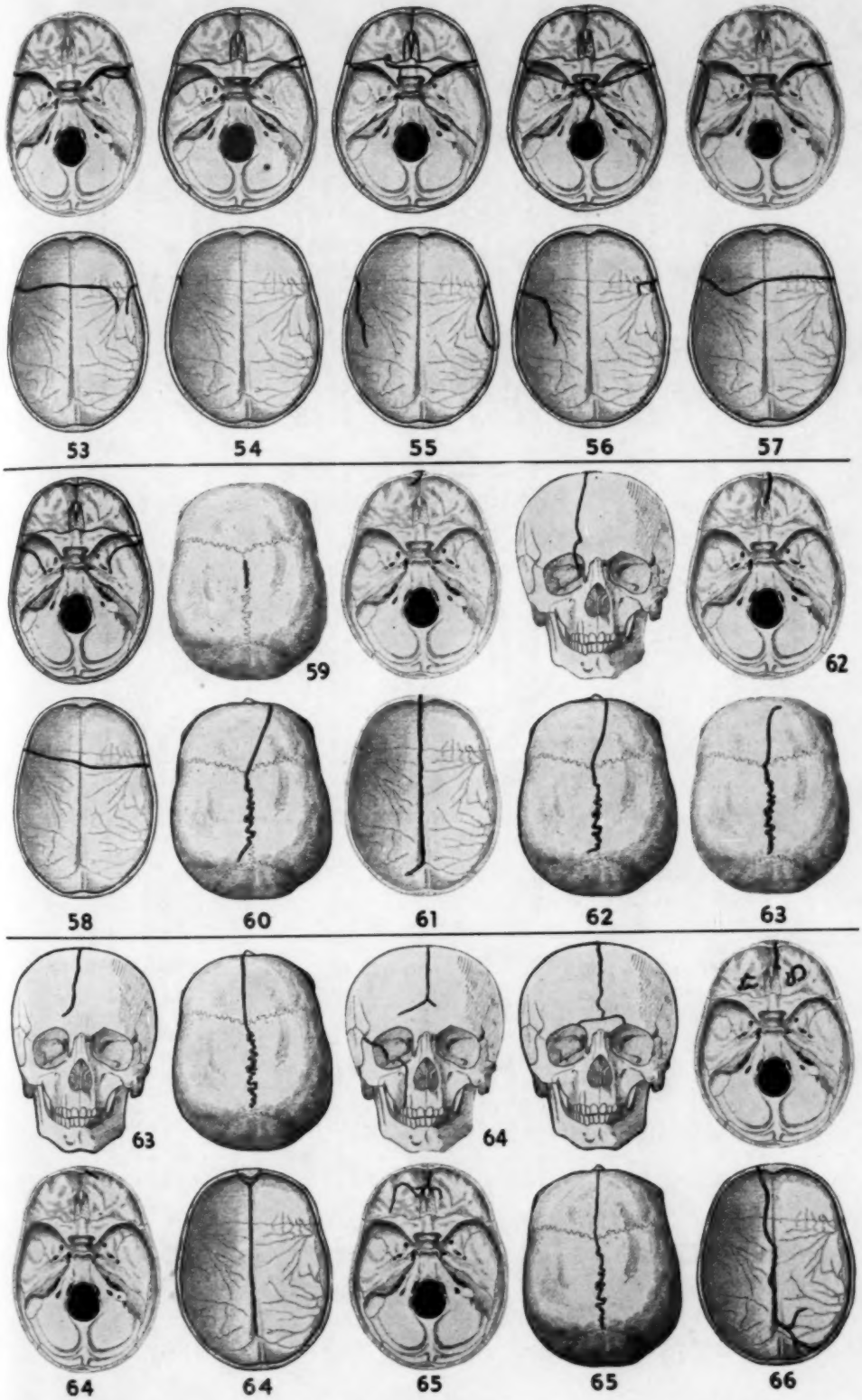

Fig. 8.—Ventral transverse incomplete fracture (*53* to *58*) and sagittal fractures of the vault (*59* to *66*). For fracture *64*, there are four figures; for both *65* and *66*, three, the third diagram of *66* being in figure 9.

TABLE 5.—*Data on Fractures in Figure 9*

No., Sex, Age	Weight, Height	External Injuries	Injuries of the Brain; Length of Fracture, etc.	Circumstances, Observations, etc.
67 ♀ 52 yrs.	130 lbs. 5 ft. 1½ in.	No external injury; large hemorrhage in the scalp on top of the head, presumably due to bleeding from the broken bone	Bruise 2 cm. deep of the frontal pole of the left cerebral hemisphere; extensive hemorrhage in the leptomeninges; dura intact; length of fracture not recorded.	Fell down a flight of stairs when drunk; admitted to hospital 16 hours later; coma; foam at the mouth; pupils inactive; eyes to the right; arms spastic; reflexes lessened or absent; in the hospital 21 hours, 45 minutes; diagnosis, uremia
68 ♂ 30 yrs.	148 lbs. 5 ft. 10½ in.	No fresh or recent external injury; large hemorrhage of practically the entire top of the scalp, bregma to inion, 19 cm. wide	Twenty grams of blood between the dura and calvarium; the top along dural sinus intact; contusions of the bottom of each frontal lobe at the tip and smaller bruises of the front end of each temporal lobe; length of fracture, 20.5 cm.	Circumstances not learned; noisy coma; pupils unequal and fixed; lower extremities and right arm rigid; all reflexes except those of the left arm absent; bloody spinal fluid; in the hospital 18 hours, 45 minutes
69 ♂ 31 yrs.	225 lbs. 6 ft. 5½ in.	A repaired surgical (?) cut 11 cm. long in the scalp across the back of the head, 12 cm. above the inion	Bruises of the under surface of the frontal lobes, 4 by 2.5 cm., with smaller bruises of the frontal lobes elsewhere; small contusions of the temporal lobes; dura intact; length of fracture not recorded	Circumstances not learned; bloody spinal fluid; in the hospital 5 hours 5 minutes, diagnosis, cerebral hemorrhage
70 ♂ 35 yrs.	119 lbs. 5 ft. 5 in.	Pale blue discoloration, 6 cm. in diameter, swollen from deep bleeding in the right half of the forehead; blood in the ethmoid cells	Deep bruises of the front poles of each frontal lobe and of the inferior surface of both frontal and both temporal lobes; one bruise of the left parietal lobe; multiple small bruises deep in the pons; blood in the lateral ventricles; the dura intact; length of fracture, 25.5 cm.	Fell off street car; coma; entire scalp of the vertex edematous; bloody spinal fluid; convulsions; froth in the mouth; in the hospital 22 hours, 5 minutes; diagnosis, skull fracture
71 ♂ 28 yrs.	160 lbs. 5 ft. 8½ in.	Bruise of the back of the head in the midline 3 cm. in diameter; blood in the left nostril; swollen purple eyelids; torn liver; right hemothorax	Bruises of the lower surfaces of both temporal lobes; dura intact; length of fracture not recorded	Fell from a viaduct; coma; bleeding from the nose and mouth; flaccid extremities; stertorous breathing; in the hospital only 30 minutes; diagnosis, skull fracture
72 ♂ 33 yrs.	98 lbs. 5 ft. 8 in.	Purple discoloration of the lids of both eyes and of the cheek adjacent to the right lower eyelid; left temporoparietal decompression operation	Deep bruise of the inferior part of the right occipital lobe of the cerebrum; fibrinopurulent leptomeningitis; superior longitudinal dural sinus intact; length of fracture, 28 cm.	Fell from an automobile when it struck a curb; decompression on the day of entrance; in the hospital 15 days; diagnosis, skull fracture

73 ♂ 30 yrs.	132½ lbs. 5 ft. 9 in.	Skin of the forehead, eyelids, of the scalp in front and behind the ears and of the right side of the neck purple from deep bleeding; right decompression operation	Bruises of the bottom of each frontal lobe; much smaller bruises of the bottom of each temporal lobe; acute fibrinopurulent meningitis; superior longitudinal dural sinus intact; length of fracture, 30.5 cm.	Found sitting on the sidewalk after a fight; delirious and could not walk alone; absent scrotal and abdominal reflexes; eye reflexes could not be obtained; involuntary urination; decompression operation the day after entrance; in the hospital 9 days; diagnosis, skull fracture
74 ♀ 65 yrs.	100 lbs. 5 ft. 1 in.	One lacerated wound 10 mm., and a second 12 mm. long, both close to the inion; a third 15 mm. long above and behind the right ear; a fourth opposite the left angle of the mandible; small bruises of the scalp; bruised right elbow	Superficial contusion with leptomeningeal hemorrhage of the entire lower surface of the left frontal lobe; similar smaller lesions of the right frontal lobe and front of the right temporal lobe; length of fracture not recorded	Circumstances not learned; in the hospital only 1½ hours; diagnosis, skull fracture
75 ♀ 38 yrs.	122 lbs. 5 ft. 6 in.	Crucial laceration of the scalp in the occiput; bruised arms, forearms, left thigh, both legs and lower lip	Extensive and deep bruises of the inferior part of each frontal lobe; extensive leptomeningeal hemorrhage; length of fracture, 21.2 cm.	Found moaning at the foot of a basement stairs; sent to the hospital of the House of Correction to be treated for alcoholism; deep coma; bloody spinal fluid; general muscular rigidity; in the hospital 40 hours; diagnosis, skull fracture
76 ♂ 35 yrs.	128 lbs. 5 ft. 6½ in.	Lacerated wound 3 cm. long just to the left of the midline of the vertex of the head	Contusions of the bottom of the frontal lobes near the midline; many contusions of the bottom of each temporal lobe, more of the left; multiple small traumatic hemorrhages in the pons; length of fracture, 26.5 cm.	Had a fit at home and fell, injuring the back of the head; convulsions; eyes deviated to the left; bloody spinal fluid; in the hospital 4 hours, 55 minutes; diagnosis, cerebral hemorrhage
77 ♂ 35 yrs.	133 lbs. 5 ft. 7 in.	Lacerated wound 2.5 cm. long and 1.5 cm. wide, 1 cm. above the inion, just to the right of the midline	Superficial bruises of the under surface of each frontal lobe, the top of the right parietal lobe and the under surface of the left temporal lobe; traumatic hemorrhages of both lenticular nuclei and the right cornu ammonis; length of fracture, 29.5 cm.	Knocked down (homicide), his head hitting a pavement; picked up unconscious; unconscious until death; in the hospital 8 hours, 15 minutes; diagnosis, skull fracture
78 ♂ 16 yrs.	62 lbs. 4 ft. 4 in.	Deep bruise of the scalp at the occiput 5 cm. in diameter; two other smaller bruises of the scalp at the vertex; huge lacerations of the right thigh, lower part of the abdomen and genitalia	Small superficial bruises of each frontal lobe at the anterior poles; two minute contusions of each side of the cerebrum; length of fracture, 19 cm.	Run over by a truck; extensive lacerations exposing the right femoral vessels; skin and superficial fascia torn from the lower part of the trunk; testicles bared; severe shock; in the hospital 1 hour, 35 minutes; diagnosis, shock and hemorrhage
79 ♂ 65 yrs.	114 lbs. 5 ft. 3 in.	Laceration 4.5 cm. long of the scalp, its lower end 7 cm. to the right of, and 4.5 cm. above, the inion (2 stitches)	Bruises 2.5 cm. in diameter of the front pole of each frontal lobe; length of fracture, 10 cm.	Slipped on ice and fell; coma; boggy scalp; unequal pupils; noisy breathing; in the hospital 10 days; diagnosis, occipital basal skull fracture
80 ♀ 35 yrs.	88 lbs. 5 ft. 3 in.	Irregular Y-shaped lacerated wound of the scalp, of the back of the head, 4 cm. long, 3 cm. to the right and 4 cm. above the horizontal level of the external auditory openings; bruised elbows and right arm and right thigh; blood in the right middle ear	Deep bruise of the front pole of the left cerebral hemisphere, a smaller one of the right; bruised under surface of the left half of the cerebellum; length of fracture, 31.6 cm.	Struck by a street car; unconscious; occasional vomiting; bloody spinal fluid; in the hospital 12 hours, 10 minutes; diagnosis, basal skull fracture

when together in the same body, but either alone suffices to indicate definitely the place where the force fracturing the skull is applied.

Persons afoot on the street, hit by street cars, automobiles and trucks, are knocked down and occasionally run over.[87] A few undoubtedly are violently thrown some distance, but they are seldom tossed up so as to come down head first and sustain scalp injuries of the vertex. On the other hand, head-first landings occur after ejection from vehicles in collision. But among the 23 fractures caused by violence applied to the back of the head, only 7 were produced by vehicular accidents. Eight persons fell. Presumably, the man assaulted (*9*) also fell. One, a woman (*29*), was hit on the head with an ax. For the remaining 6, the circumstances were not learned. So there is nothing uniform in the nature of the accidents. There are other factors, however, that presumably prevailed in all 23 casualties. The persons were alive when injured, with their heads, necks and trunks normally held together, their cranial cavities full of brain, cerebrospinal fluid and meninges, and all the intracranial blood vessels contained more or less blood. The head, when hit from behind, was not sent rolling away from the body. The neck held it fast, and at the lower end of the neck the weight of the trunk and extremities resisted.

Among those persons knocked down so as to fall backward, the backs of some presumably struck first, and the head, with increased centrifugal speed, struck immediately after. For others, these events may have been reversed, the head hitting first. In any case, the force applied to the back of the head in the midline was disposed, in order to make the ring fractures, either to drive the head down on the spine or to tear it away. That the latter deserves consideration is indicated by the impossibility, without fractures being produced somewhere, of forcing all the bony parts in the midline behind, from the occipital boss down to the level of the scapular spines, to lie in contact with a flat surface at the same time. In not one of the 23 ring fractures now under consideration was the neck broken. The cervical vertebrae held together, but the floor of the skull gave way to a force which, according to this view, was inclined to jolt the head away from the spine. Moreover, the floor of the skull broke where it also fractures when compressed vertex to spine or vice versa, that is, in its weakest places. When Quenu and Tesson [66] made the large ring fracture already referred to, an entire dead body was fastened to one of two heavy planks, but the plank extended only to the shoulders; the head was left free. The planks were fastened together end to end by a hinge. From an erect position, the body with its plank was forcibly thrown to the floor so that the

87. Bacon, L. H., and Le Count, E. R.: Automobile Injuries: A Study from Records of Postmortem Examinations, Arch. Surg. **18**:769 (March) 1929.

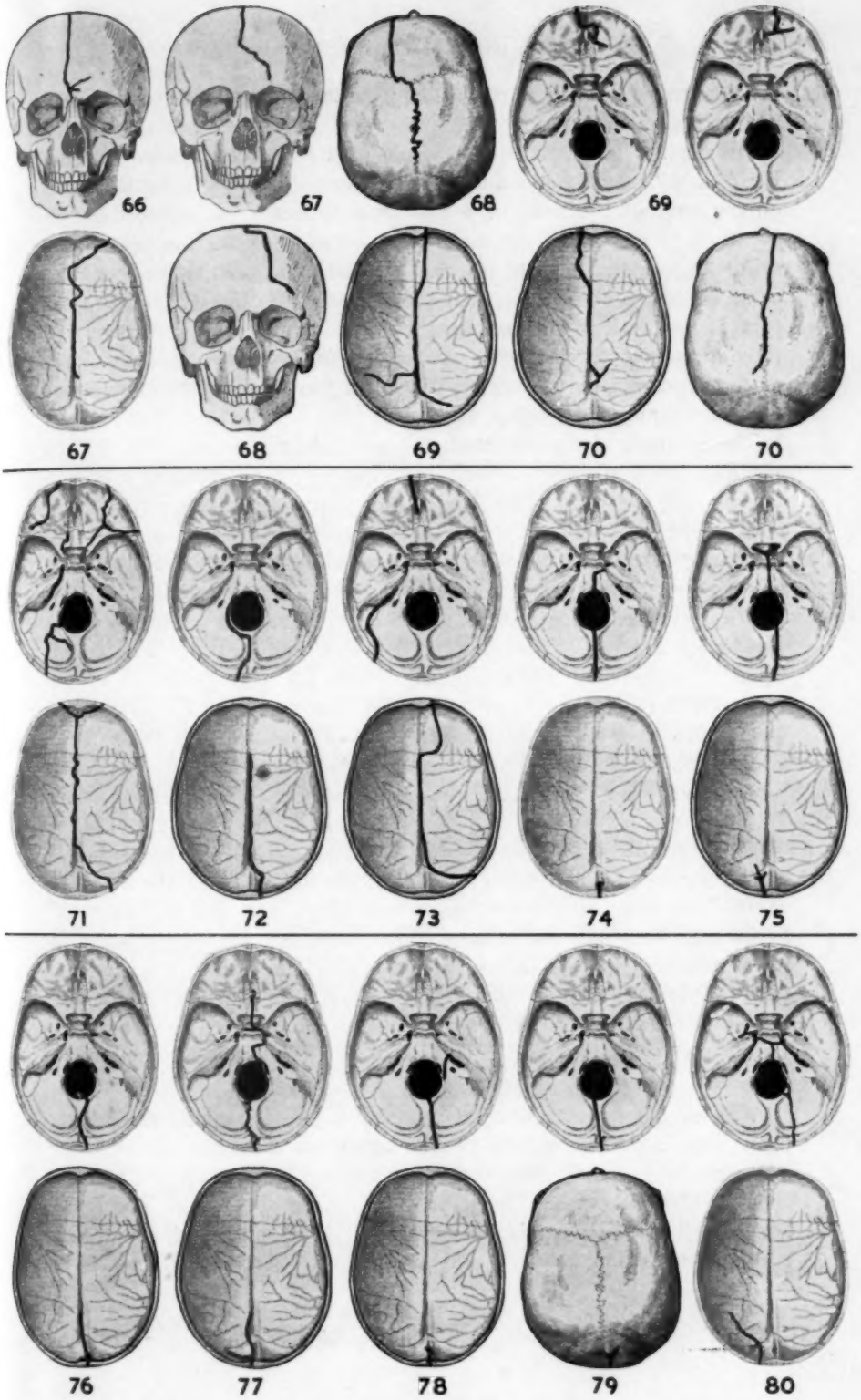

Fig. 9.—Sagittal fractures. There are three diagrams for fracture *70*.

occiput struck the rim of a curbing; the large ring fracture produced had no branches.

In another place, mention is made of a ring fracture described by Bell [88] as a "pure instance of counter fissure," the blow being delivered on the back of the head of a man in a tunnel by a bucket that fell 50 feet (15 meters). One may well wonder what the position of the man was, how far forward his head was bent and whether he was erect or on his knees, so that the falling bucket was able to hit "the strongest part of the skull in the convexity of the occipital bone." Answers to these questions correlate with whether the bucket drove the head down on the spine or partly tore most of the head away from the spine when the ring fracture was made.

There remain for consideration 4 small ring fractures (*22, 23, 26* and *35*). Two of these (*22* and *35*) probably were fractures produced by crushing the head from side to side. In 1 case both sides of the head of the boy (*22*) were bruised severely in front of each ear. The prevailing transverse course of the fractures indicates that the head was run over. Both petrous bones were loose. Opposite the transverse fracture of the basilar plate of the occipital bone, the brain stem was *completely severed*. The slightly damaged skull cap is astounding when compared with the comminuted base. A second allusion to the symmetrical ring in the front half of the base of the skull is warranted on account of its uniqueness. With the other accident (*35*) came a circumstantial tale of the body being crushed between the wheel of a wagon and a barn door, and the head was the only injured portion. In this skull, the basilar plate was also fractured across well forward.

The third of these 4 fractures occurred when the front of the thorax of the woman (*23*) was apparently hit by the street car, for the fractures of the ribs were all close to the costal cartilages. She probably fell backward. Although the brain was not contused, the hemorrhages in the leptomeninges were on the frontal lobes. The fourth fracture was due to violence behind the left ear (*26*). A bruise there was the only external injury of the head; the asymmetrical enclosure of the foramen magnum by fissures is only a detail of severe comminution spreading from the site of trauma.

Ventral Ring Fractures of the Vertex (*5*).—Apparently these fractures have not received any special attention. Descriptions of them are exceedingly rare. One interesting account deals with the popular Duke of Orleans, son of Louis Philippe I, King of France. The Duke was thrown from a carriage on July 13, 1842, and died about five hours later.[89] The cranium was broken into upper front and lower

88. Bell, p. 467.

89. Editorial, Lancette française, Gaz. d. hôp. **15**:433, 1842.

back portions by fractures on each side from the lambdoid suture forward and down in the middle fossae. The body of the sphenoid bone was broken across; the squamous part of the left temporal bone was loose and held in place only by soft tissues. Although other explanations were suggested,[90] Marchal (de Calvi) insisted that the Duke had fallen so that the back of his head on the left side first hit the roadway. In support of his contention, Marchal appeared before the Academy of Medicine [91] with a skull in which he had produced injuries similar to those in the head of the Duke. He had taken a cadaver and stood it erect on an amphitheater table. Facing it, he took it by the shoulders and hurled it to the floor so that the back of the head on the left side struck first. He described the resulting fracture as follows:

> Cette fracture, qui s'étend de l'occipital, à droit et à gauche, jusqu'à la base du crâne en passant par le milieu de la selle turcique, et qui divise le crâne en deux moitiés, l'un inférieur et postérieur, l'autre antérieur et supérieur est semble à celle du prince.

His demonstration created great interest at the Academy and is set forth here at length because, although the fractures in the Duke's head are referred to frequently in the older literature, their experimental duplication by Marchal has been generally neglected.[92] In both skulls there were ventral ring fractures of the vault, but the upper front portions broken away from the rest of each cranium, judging from Marchal's descriptions, were somewhat larger than similar portions in craniums 38 to 42, inclusive. Berchon [93] described a healed fracture that ran all the way around the top of the skull horizontally, but lower on the left side, below the occiput behind and through the left frontal eminence. Berchon found the skull in the museum at Rochefort. It was brought there from New Caledonia. In the cranium of a boy who died six months after a bicycle collision, Phelps [94] noted a healed fracture which extended "from the left temporal fossa across the forehead, three-fourths of an inch above the supraorbital ridge, to a point three-fourths of an inch internal to the right external angular process and then with a curve passed upward and inward to the median point of the

90. See the letters by Le Gros and Tanchou,[89] pp. 406 and 407.

91. Footnote 89, p. 432; see also: Marchal (de Calvi): Relation chirurgicale de la morte du prince royal, Ann. de chir. franç. et étrang. **5**:465, 1842.

92. Marchal was evidently present in the little shop where the Duke died. His account of what transpired and mention of those present is touching and circumstantial. Guizot in the last volume of his "History of France" also described the distressing scene and said he was there three hours.

93. Berchon, E.: Fracture circonscrivant presque régulièrement la voute du crâne d'un sauvage de la nouvelle calédonie, cicatrisation osseuse complète, Bull. Soc. anat. de Paris, series 2, **8**:95, 1863.

94. Phelps,[19b] case 117, p. 466; case 51, p. 466, and case 82, p. 444.

coronal suture." The description he gave of the fractures in two other skulls also suggest ventral ring fractures of the vault. But there are no illustrations, unfortunately, and connected with the fractures of the vault there were other fissures radiating down in the lower half of the cranium. In the long analysis of skull fractures by Brun [2b] there are illustrations of the course followed in 127 skulls by fractures of the type from which our 80 were chosen, but none is a ventral ring fracture of the vertex. Other contributions like this by Brun have been examined with similar results.

The first 3 of the ventral ring fractures of the vertex in our series (*38* to *42*, inclusive) were fairly symmetrical; the other *2* were decidedly less so. There were bruises and small lacerations of the left side of the face of one of these 5 bodies (*38*). The lower left incisor was freshly knocked out, the corresponding upper tooth loosened and the tongue torn. Directed obliquely across the head was a lacerated wound behind the right ear. The brain injuries were mainly of the bottom portion and in the frontal and temporal lobes. The front cerebral tips were not contused. The nature of these injuries together with the fracture-dislocation between the fifth and sixth cervical vertebrae indicates that the face was hit first and the man knocked backward; also that the head was in motion when it was arrested. Although the force breaking the skull was obliquely directed from behind forward and from right to left, the symmetry of the cup-shaped disk almost broken from the rest of the skull is remarkable. In the next head in this group (*39*), there were also contusions and small fresh tears of the face, mainly on the left side, but no other external injury of the head. There was a large bruise in front of the left ear on a third head (*41*), with a lacerated wound in the scalp covering the front of the right frontal fossa. If these injuries were produced by a side to side compression because the taxicab ran over the head, they probably were made after the skull was fractured, for the 3 fractures radiating forward from the lambda are unlike those produced by violence applied to the sides of the head. In the soft tissues of the other 2 heads (*40* and *42*), there were no external injuries except a curved surgical incision behind the right ear of 1 (*42*). Close to the bone blood had spread from the fractures in the deep scalp tissues of both heads and in the orbits and eyelids of 1 (*40*) where the floor of the frontal fossa was broken extensively. None of these 5 ring fractures was complete. In 4, the gap was at the front end; in the fifth (*40*), it was in the vertex. The bottom of each of the 5 brains bore signs of injuries in the front half; in 1 (*39*) there was simply leptomeningeal hemorrhage; in the others, contusions as well. There were no contusions at the front of the cerebrum in any of the 5 brains. Four of the fractures were caused

by motor vehicles. How the fifth (*42*) happened was not learned: it probably was a similar accident. "Alcoholism and suspected skull fracture" was the only recorded diagnosis.

The similarity of these fractures implies a force acting in the same direction for all 5. One of the bodies (*42*) came to necropsy with the head entirely shaved. Tops of 3 other heads were bald (*38, 39* and *41*). Under such conditions, had there been injuries of the outer layers of the scalp opposite the dorsal parts of the ring fractures, they would not have escaped notice. A fourth head (*40*) was that of a Mexican with coarse black hair 15.5 cm. long on top. The fractures in this cranium differed from those in the other 4. They were unconnected in the vault and comminuted in front. The natural inference that violence was applied to the face has no support from injuries there, for there was none. The eyelids were purple from bleeding which developed gradually in the hospital. Moreover, at the time of entrance there was "a lump over the right parietal bone." In the scalp of the fifth head (*38*), there was a lacerated wound on the right of the occiput, but the bruises of the face, as well as 3 broken ribs, were on the left side. Apparently the automobile struck the man in front, but more on the left side, knocking him down, and when the head hit the road the symmetrical ring fracture of the front portion of the vault was produced.

It is difficult to exaggerate the importance of the location of the brain injuries in any venture to understand how these 5 fractures were produced. Some brains bore bruises elsewhere, but the front half of the bottom of each was injured. The front tips of the cerebral hemispheres were uniformly unhurt grossly. These conditions have two definite implications: that the heads were in motion and that abrupt arrest and fracturing occurred when the back of the crowns struck something. One natural corollary is that 3 of the 5 heads, and possibly a fourth, struck soft or padded surfaces, and the scalp opposite the injured portion of the brain escaped injury.

In our review of the literature on ring fractures of the base of the skull there is abundant evidence of interest in the influence that the weight and the resistance of the trunk have in the production of the fractures. But the part played by the trunk mass in causing other varieties of cranial fractures has not received the attention it merits. A far greater and reprehensible neglect has been the failure to emphasize properly the importance of the face mass. In both particulars this disregard has been only relative.[95] We have been unable to find measure-

95. Duret: Sur le mécanisme des fractures du crâne, Arch. gén. de chir. **6**:689, 1910.

ments of the weight of the face mass. The mandible, other bones of the face and the soft parts of the face that we removed from one cranium soon after death weighed 815 Gm., but they were well drained of blood.[96] During life they probably weighed at least 2 pounds (0.9 Kg.). With the increasing momentum such a mass has at the end of a backward fall, its pressure on the bottom of the front end of the cranium must be considerable. If before the fall the body is thrown upward somewhat and in falling comes down head first and obliquely to the ground, as an arrow does at the end of its flight in target shooting, and the back part of the crown strikes first, conditions exist which may account for these ventral ring fractures of the vertex (*38* to *42,* inclusive). In these 5 skulls the parts below the ring fracture held fast, whereas the face mass tended to move on with the cranium above the fracture. Their momentum was not checked simultaneously with the rest of the head. The force was delivered obliquely near the occiput and in the midline, and the resulting ring fractures are obliquely and symmetrically disposed in the front end of the cranium. This explanation has some support from the presence of broken necks in 2 of the 5 bodies (*38* and *41*). With the other 75 fractures in our series, there were only 2 other broken necks (*14* and *32*).

Symmetrical Fractures of the Orbital Roofs (6).—In searching for accounts of ring fractures, we came across many records of asymmetrical independent and solitary fractures of the base of the skull. Their inclusion in a table, because of the historical value it would possess, was seriously entertained. But the number of such fractures observed is too large and, although intimately related, they are unlike our 80. We were able to foresee that in this omitted table there would be many short segments of ring fractures. Among about 90 of these asymmetrical fractures of the base of the skull we found 22 solitary or independent fractures in the petrous bones and 22 in the orbital roofs. Fourteen of the latter were on the right side and 4 on the left, and for 4 the side was not stated. We also found 11 reports of more or less symmetrical fractures in both orbital roofs with, in 9 of the skulls, fractures elsewhere in the cranium. In the 2 remaining,[97] the fractures were solely in the orbital roofs. This we encountered in our case 32; associated injuries were bruises of the nose and forehead and a fractured third cervical vertebra. The other symmetrical fractures of orbital roofs (*2, 9, 22, 52* and *66*) were associated, but unconnected, with fractures elsewhere in the cranium.

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96. The body was not embalmed.

97. Leriche: Fracture du crâne; épanchement de sang à la base; kyste de l'arachnoide, Bull. Soc. anat. de Paris, series 1, **10**:55, 1835. Vincent, E.: Contribution à l'étude des fractures indirectes de la base du crâne et des lésions consécutives aux traumatismes cérébraux, Alger méd. **15**:141, 1887, case III.

The face mass probably had considerable influence in causing some of these. In 2 heads with fractures from falls (*2* and *9*), there were large ring fractures with contrecoup bruises of the front end of the brain. The fractures in *66* were also from a fall; they were sagittal in the vault, with the lower surface of the brain bruised in front. These 3 heads (*2, 9* and *66*) were in motion when broken. The vault of a fourth head (*52*) was hit by a hammer on the left side. The fracture was transverse, and the part enacted by the face mass was one of resistance, such as the spine exerts at the other end of the base of the skull. It has long been known that solitary fractures of the orbital roofs often are caused by blows on the forehead or elsewhere on the upper part of the face. Bouchacourt,[98] as long ago as 1840, in discussing a presentation by Tavignot, commented on the facility with which this is accomplished on cadavers. The unevenness of the orbital plates causes them to buckle when axes are shortened.[99] Small fragments of thin, translucent bone remain attached to the dura when the bone is bared, and tags of orbital fat are forced in the cranial cavity. Violence of this sort caused the fractures in case 32.

Although it is always surprising to find the entire cranium or only its upper or lower halves split in two parts by either sagittal or more or less symmetrical transverse fractures, the experience is fairly common in postmortem examinations for medicolegal purposes. The fractures usually have direct paths easily reconciled with both the direction of the causal force and the site of its application. For these reasons, the attention given these fractures in medical literature is inconsiderable and devoted chiefly to recording their incidence. In this special group of 80 selected fractures, 38 are either transverse or sagittal. We shall restrict our discussion to the evidence obtained about the nature of the violence causing them, the way it acted and the degree with which this violence is reflected in the peculiarities of the fractures.

Dorsal Transverse Fractures (10).—The first 4 fractures in this group (*43* to *46*, inclusive) were in the base of the skull and were only about half way around. They extended so short a distance in the skull cap on each side that the sketches are not reproduced. In the next 3 skulls (*47*, *48* and *49*), the fractures run a little higher, and their location is represented on diagrams of the sides of the skulls. The dotted lines represent the course on the opposite side. Two skulls (*50* and *52*) were broken almost in two, and 1 (*51*) was completely broken. In 3 (*44*, *48* and *49*), the fracture ran across the front of the clivus.

98. Bouchacourt, in discussion on Tavignot: Observation de fracture du crâne et de contusion du cerveau, Bull. Soc. anat. de Paris, series 1, **15**:37, 1840.

99. This inbending was demonstrated experimentally by Chipault and Braquehaye.[35]

There is no need to recapitulate what has been written about fractures of this kind. They are not uncommon, and it has been quite generally accepted for many years that they are caused by force applied to the sides of the skull. The manner in which 2 of the 10 fractures (*43* and *49*) were sustained was not learned. One (*45*) was due to a fall; another (*50*), to being hit by a street car; another (*52*) resulted from slugging by burglars, and motor vehicles caused the remaining 5 (*44, 46, 47, 48* and *51*).

Our interest concerns the external injuries and the injuries of the brain and the bearing that their location has on the character of the fracture. The information about 1 body (*43*) is too defective, and no external injuries were present on a second (*45*), except a bruise of the back of the right elbow. Such bruises are very common on the bodies of persons who fall backward or who are knocked down. Some of the bruises of the brain of this body (*45*) were at the front end. It was said this man fell downstairs. His head may have hit more than once.

Interpretation of what happened when 3 other bodies were injured is beset with difficulties. One of these (*47*) was the body of a man 6 feet tall (182.9 cm.), weighing more than 200 pounds (90.7 Kg.). A lacerated wound was found in the right temple; there were 11 fractures in 7 ribs on the right side; the right innominate bone was broken in its pubic and ischiatic portions, and the left pubic bone was broken close to the midline. The right side of the forehead and right eyebrow were bruised. The most reasonable explanation is that when the man was hit he fell forward, bruising the face and the back (contrecoup) of the brain; then he was run over so that the ribs, pelvic bones and skull were fractured. The alternative is that all the injuries of the right side, including the fractures, were first received when he was struck by the automobile, and that subsequently a fall forward injured the face and brain. The automobile that another man (*51*) was driving at a high rate of speed collided with a truck. In the pons, there were multiple minute hemorrhages indicating severe impact. This is supported by the loosening of a fragment of the calvarium above and behind the right ear. Another possibility is that when the skull broke, the front and back fragments bent a little, the one on the other, for the fracture ran all the way around, and that when bending occurred, the pons was bruised. But scattered small hemorrhages are frequent in the pons from trauma [100] when the skull is not broken in two, and they accompany fractures which fail to correspond to any special pattern. Absence of injury of the left hemisphere indicates that the head was not moving

100. In this connection see the article by Greenacre, P.: Multiple Spontaneous Intracerebral Hemorrhages, Bull. Johns Hopkins Hosp. **28**:86, 1917.

at any considerable rate of speed from left to right when the blow was received that broke out the piece behind the right ear. The left cheek was bruised, and this, with the absence of contrecoup bruising of the brain, points to a side-to-side compression of the head.

As for the third body (*52*), a Negro whose head was hit on the left side with a hammer by burglars, the sole question concerns bruises antipodal to the region of comminution in the left side of the skull cap. The outside of each temporal lobe and the bottom of both the frontal and the temporal lobes were bruised, the bruises on the right side being much larger than those on the left. It would seem, therefore, that after the man was slugged, he fell so that the left side of the head struck. This assumption involves the possibility of a localized fracture of the left side of the head made with the hammer, and a transverse fracture, almost complete, due to the fall. Injuries in the other 5 bodies (*44*, *46*, *48*, *49* and *50*) are accounted for easily. Brain injuries were more marked or limited to the right side in 4 bodies (*44*, *48*, *49* and *50*), and there were external injuries of the left side of each head. In the fifth body (*46*), these conditions were reversed. In other words, both the external and the cerebral wounds corroborate trauma to one side of the head.

Transverse Ventral Fractures (6).—It seems that these sidewise circular fractures prevail in the bottom half of the skull. Of the 10 dorsal fractures just reviewed, 7 were incomplete in the vertex and 2 in the bottom. Of the 6 ventral sidewise fractures, 4 had their gaps in the skull cap (*53*, *54*, *55* and *56*); 1 was unfinished in the bottom (*58*), and 1 was probably complete (*57*). Fracture *53* was almost complete. Of particular interest is the sagittally directed fracture in 1 head (*56*) from the sella to the front rim of the foramen magnum. With 4 of these 6 ventral ring fractures (*53*, *54*, *56* and *57*), there were injuries of the outside of the head on one side, usually farther forward than those accompanying the dorsal transverse fractures. In each of these 4, there was sufficient bruising of the brain opposite the external wounds of the head to stamp it as indirect. These 4 heads were in motion when struck, and the movement was in paths connecting their external injuries with the opposite brain contusions. One of these bodies was that of a man who, it was said, was run over by a horse and wagon (*54*). His injuries conform simply to those inflicted by being struck down. There were tales of falls concerning 2 other cases: in 1 the fall was over a stair railing (*57*) and in the other, from a porch on a third floor (*56*). In the latter body, the injuries of the trunk were severe.

In another instance it was said that the woman (*55*) was "run down by an auto." Apparently, the fractures of the cranium and mandible were

produced when the wheel of the machine passed over her head, for the brain bruises were on the right side where there was a huge abrasion of the face; the fracture in the lower jaw was opposite. The rarity of this grouping of injuries of the head and brain is worthy of comment. The last of these 6 ventral crosswise fractures (*58*) is of even greater interest, because it was made with an ax. A portion of the fracture was a cut with some small fragments of bone at its site.[101] One of the fragments had been driven into the deep wound in the top of the brain. One remarkable feature of the injury of the head was that the long dimension of the edge of the ax was directly across the top of the head.

Sagittal Fractures (22).—The remaining fractures in our series of 80 are mainly or altogether in the sagittal plane. The symmetry is of fragments, not of fracture lines. The first 12 (*59* to *70*, inclusive) were mainly or altogether in the vault; the next 3 (*71*, *72* and *73*) were in the vault and base, and the rest (*74* to *80*, inclusive) were exceedingly basilar. Judging solely from the inner surface of 1 cranium (*66*), there were independent fractures in the orbital roofs; in another (*64*), the bones of the face were broken on the right side; lengthwise fractures of the clivus were present in 4 (*74*, *75*, *77* and *80*); in 4 heads (*61*, *63*, *72* and *73*), there were unilateral surgical decompression wounds of the scalp and cranium. A history of a fall is quite common with sagittal fractures. One man (*70*), it was said, fell from a street car; another (*77*) was knocked down in a fight; a third (*73*) was found sitting on a sidewalk after a fight; 1 of the 4 women (*75*) was found moaning at the foot of a stairway; for 7 others (*60*, *65*, *66*, *67*, *71*, *76* and *79*) the history of a fall was even more definite and reliable. This makes a total of 11 fractures due to falls. Six of the remaining 11 (*61*, *62*, *63*, *64*, *72* and *78*) belong under the caption of accidents from motor vehicles; another (*80*) fracture resulted from being hit by a street car, and the circumstances for 4 (*59*, *68*, *69* and *74*) were not learned.

At present, there seems to be no reason for dealing separately with these sagittal fractures, because some were basilar and others were in the dome. In fact, comments on the mechanism of their production follow rather simple patterns. With the first (*59*), there was no scalp or brain injury; the blood in the scalp was from torn intra-osseous vessels, and there was no history of injury. Accompanying another fracture (*65*), there was no brain injury, but the leptomeninges on the vertex of the cerebrum were livid from traumatic bleeding. There

101. These fragments were so small that no attempt has been made to represent them in fracture *58* (fig. 8).

are, consequently, for these 2 fractures no reliable indications of where violence met the head. Limitation, or preponderance of, leptomeningeal hemorrhage and torn brain tissue to the pole of the head where there was external injury, coupled with absence of injury of the scalp at the opposite pole, are conditions associated with 4 other fractures (*60*, *61*, *62* and *72*); such brain contusions are direct, and blows to these heads were probably close to the end of the fracture nearest the damaged brain. There were signs of trauma at, or not far from, the vertex of 3 other heads (*66*, *71* and *76*) with contrecoup bruising of the bottom of each brain, more toward the rear than usual. These were in the bodies of 3 men who fell; it seems fair to assume that the craniums were not previously freshly fractured. In 3 more heads (*67*, *68* and *70*), the brain wounds probably were contrecoup notwithstanding absence of external injuries at the opposite pole. No information was obtained of how the injuries of 1 body (*68*) were sustained, but falls caused those of the others (*67* and *70*). This disposes of 12 of the 22 sagittal fractures: 3 with force to the top of the head and indirect bruising of the bottom of each brain with the heads in motion when injured; 4 associated with direct brain injuries and 3, with brain injuries, probably contrecoup; for 2, no assumptions are warranted. It is remarkable, but not at all astonishing, that with the other 10 fractures there were uniformly contusions of the brain at one pole of the sagittal fracture and scalp injuries at the other, or, in some instances, at both poles. In other words, the craniums were broken when their motion was checked suddenly, and fractures beginning at the pole where the blow was received coursed sagittally from that point.

When the medicolegal postmortem examinations resulting in our records of the 1,278 linear traumatic cranial fractures began in 1911, interest in satisfactory explanations of the fractures was not so acute as during subsequent years. From the onset, however, when spots of hemorrhage were seen in the deep layers of the scalp, cuts were made to learn how close to the external surface the bleeding extended. Decisions were made as to whether the blood was from torn vessels in bruised layers of the scalp or from vessels within or close to the bone torn because the bone broke. Tears in small blood vessels in the fat just beneath the corium of the scalp occur sometimes without abrasions, discolorrations or other signs of trauma on the surface. In many scalps, especially those of Negroes, this adipose tissue is a thick layer and can easily accommodate considerable blood. With death soon after accidents, the blood accumulating in this fat is less and the time is too brief for its infiltration to the deep layers, where it is promptly noticed when the scalp is turned inside out in front and back flaps on the

cranium. Appreciation of the significance of these conditions gradually increased in the course of time, so that in the latter part of the period, from 1911 to 1924, cuts from within outward were often made in the scalp at places antipodal to brain injuries when the deeper scalp layers bore no stain of blood. Naturally, in this way, some scalp contusions previously unsuspected were discovered. Applications of the principles involved also brought about detection of healed cranial fractures diametrically across from superficial *plaques jaunes* of the brain. Some of these healed fractures were barely visible, and then for only a portion of what was undoubtedly their original length and distribution. It is probable that had the methods of examination of the scalp been thorough throughout the period of thirteen and a quarter years, scalp wounds squarely across from brain injuries would have been found with more than 10 of the 22 sagittal fractures.

MECHANSIM

In the comments on the 80 fractures in our series and their concurrent injuries there are many allusions to the way the fractures were made. Ample consideration has been given independent fractures of the orbital roofs and ventral ring fractures of the vertex. In general, for fractures with satisfactory information about wounds of the scalp, brain and other parts of the body, as well as the circumstances attending the casualties, one of the main justifiable conclusions is that the directions of the force and fracture are usually parallel. Questions of priority for any of the accepted theories of the mechanism of traumatic fractures of the cranium are difficult to settle. Theories and their discussion have already endured two thousand years. But there is unwonted accord by modern writers in giving credit to Schwartz [17] for establishing this conformity in the direction of force and violence. However, it is quite likely, as their investigations were published only a year apart, that while Schwartz was arriving at his decision by studies of fractures from human calamities, Félizet [15] was obtaining a similar inference with a hammer and chisel in experiments on human skulls. The homicidal fracture made with an ax (*58* in our series) is singularly corroborative of Félizet's results. Other transverse fractures and many sagittal fractures in our series are indubitable vouchers for this agreement between violence-direction and fracture-direction.

From the place struck squarely with blunt force, fractures should radiate in all directions as meridians toward the opposite pole. All such meridians lie in planes along which violence is propelled. It is true that from some places in the vault where the thickness of the wall is fairly uniform fractures do radiate in various meridians, for-

ward, backward, down the sides or at other angles, but those passing down as transverse fractures are more numerous than fissures with an oblique or sagittal course. This is brought about by a number of factors, among which are resistance of the spine behind, the inertia of the face mass in front, the thin lateral walls and the protrusion of the base toward the center of the cranial cavity at the transverse spheno-occipital synchondrosis. Meyer [102] called attention to the spiral contour of the periphery of the cranium in the sagittal plane—a smooth curve up and back from the glabella and over the vertex continued as a smaller coil from the occiput down, then forward and, finally, upward in the clivus. This also promotes an excess of transverse folding of the cranial floor, so that the fractures yield front and back more than other fragments. As was pointed out in the discussion of the transverse fractures in our series, they are of two sorts: dorsal and ventral. This is well shown in the composite illustrations by Félizet [103] and Dulles,[104] in which the paths followed in the inner table of the bottom half of the cranium by many fractures are represented in single diagrams. The region of greatest exemption in the floor of the middle fossa is that formed by the large wings of the sphenoid bone. Although the bone forming them is thin and usually translucent, they are protected outside by the thick crest dividing the zygomatic and temporal fossae, and to the rear of this by the heavy ridges of the roots of origin of the zygomatic arches. On the inside of each greater wing, the pterygoid processes jut forth vertically downward from the outer surface and act as girders for the floor. Fractures cross the base in front and behind these processes, but rarely through them. Behind them, the large carotid foramina and in front, the sphenoid clefts and optic foramina, seem to act as lures for the breaks.

But violence is more commonly below the crown at or near the base level.[105] This vaguely defined horizontal belt in the cranial wall is quite plain in front where the junction of vault and floor approaches a right angle. Passing back, its distinctness lessens and is only faintly evident in the rounded wall where the hemispheres of the cerebellum lie. Radiation of fractures from the base level and from places just above or below this level is limited by architectonic features more profoundly than is the case with those beginning higher in the vault.

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102. Meyer, G. H.: Die Statik und Mechanik des menschlichen Knochengerüstes, Leipzig, Wilhelm Engelmann, 1873, p. 236.

103. Félizet,[15] plate 1, p. 44.

104. Dulles,[10b] plate XXVIII, p. 337.

105. Baum, W.: Beitrag zur Lehre von den indirekten Schädelfrakturen, Arch. f. klin. Chir. **19**:381, 1876. Rawling, L. B.: Fractures of the Skull, Lancet **1**: 973, 1904. Walton.[18]

For the buttresses [106] running up and down from the base level hinder horizontal bending. The panels between the buttresses behave like a barrel stave does when, lying convex side up, it is jumped on; it breaks across. Fractures run vertically in the panels because the buttresses do not spread apart readily. They support the panels horizontally as the ground supports the ends of the barrel stave. With sufficient force, of course, the buttresses also break. But radiation is usually up and down in these regions, and fractures are guided by the buttresses into portions of the cranial wall between them. Although others, notably Aran [12] and Trélat,[6b] had observed the frequency of vertical fractures in the panels, Félizet's prestige for the explanation of their course has been and probably will continue to be time-honored. At this point it is expedient to add to the factors conducive to transverse radiation from the vault the greater distance between the two lateral, than between the other, buttresses.

The accounts of the 45 ring fractures in the literature are full of statements about headlong falls, and exceptionally about falls so that the bottom of the cranium was violently pushed up by the spine. These, supplemented by the experimental production of a few ring fractures made by blows on the vertex, leave the impression that basal ring fractures are due to force vertically directed from above or below. Such an impression clashes with the profusion of evidence that many of our 36 basal ring fractures were produced by violence applied at the occiput. But this apparent disagreement should not be taken seriously, because the fractures in the two series are so similar that it is fair to assume that rather uniform conditions prevailed in the entire lot of 81 basal ring fractures. There were several mechanisms. Excluding those made experimentally, the incidence of the different mechanisms in the two series, in all likelihood, was much the same. A few were due to impact of the spine, some from force directed down from the vertex and many from bumps on the back of the head. The way force alined with the spine makes these ring fractures requires no explanation.

It may be recalled that the 80 fractures represented in figures 5 to 9 were selected from the records of 1,278 linear cranial fractures due to blunt force. With a view to better understanding of the basilar ring fractures from injury of the back of the head, a cross-section

106. In the accounts of skull fractures most textbooks give some consideration to these thicker vertical parts of the cranial wall. Trélat,[6b] in 1855, and subsequently Félizet,[15] in 1873, called them buttresses ("poutres du crâne") of Rathke. Hilton also described them. "Arcs-boutant" and "murs-boutant" are other designations (Duret [96]). They number 6: 1 in front and 1 behind, both in the sagittal plane; 1 on each side with the external angular process as its strongest part; a second lateral pair including the mastoid processes and petrous bones. See Le Count and Apfelbach.[4a]

study was made of the remaining 1,198 necropsy records to learn what sort of fractures result from blows at the occiput when ring fractures are *not* produced. The records examined numbered 352, about 30 per cent of the 1,198. They concerned necropsies made in six short periods quite evenly spaced during the thirteen and a quarter years. Of these 352 linear cranial fractures, 40 were produced by force at or near the occiput; each one of the 40 was associated with wounds of the scalp covering the lambda or the inion. Among the 40, there were 10 sagittal fractures behind the foramen magnum with more or less irregular branching at their front ends; 2 others were paramedian.[66] The remaining 28 of the 40 fractures were rings or segments of either large or small rings. Two were ring fractures with so much asymmetry they were not used to augment the selected 80. A third fracture was simply the back half of a large ring; another coursed horizontally in the back half of the cranium; 3 followed the lambdoid suture to the left, and 3 others to the right, from the midline. The rest of the 28, as one would expect, were fractures lengthwise the occipital panels: 8 were in the left panel and 7 in the right; and in 3 craniums both panels were split with fractures beginning at the inion. From this reckoning, 11, or 12 per cent, of the 1,278 fractures were from injury of the back of the head, and the fractures so produced were roughly one third in the midline and two thirds in rings or segments of rings.

A more interesting outcome of the survey is the conclusion that both large and small basilar symmetrical ring fractures produced by trauma at the occiput are simply part of a pattern which includes sagittal and paramedian fractures coursing forward in the base and many fractures which may be regarded as segments of rings. The suggestion has been made[107] that force at the occiput is transmitted directly to the top of the spine. This opinion is based on the oblique position the cranium has on the top of the spine, with the front of its floor higher than the rear and with the anterior portions of the opposed articulating surfaces of the atlas and occipital condyles markedly sloping upward and forward. Force directed obliquely against an immovable, irrefrangible object tends to ricochet at an angle equal to that of collision. Instead of driving the skull down on the spine, according to this view, force from the occiput is disposed to tear it away from the top of the spine. But the atlas is so firmly bound to the occipital bone that they do not tear apart. And so it happens that portions of one or both lateral halves of the cranial floor are broken away from the remainder by sagittal or paramedian fractures. On rare

107. Masse, E., and Cam, St. Pierre: Etude médico-légale à propos d'une fracture du crâne par un coup du baton sur le vertex, Ann. d'hyg. pub. et de méd. lég., series 2, **37**:124, 1872.

occasions, the parts left attached to the spine are more or less symmetrical cup-shaped disks with diameters which vary widely. Most frequently, the fractures run for only long or short distances in locations where such disks have their rims. This suggestion to explain ring fractures of the base from force directed against the back of the head has some confirmation in the fracture produced experimentally by Quénu and Tesson.[66] Another, but quite different, fracture from violence at the occiput, made by Perrin,[108] is cited by many writers (fig. 10). It was made by dropping the head of the body of a young man, on a padded surface. In his comments about this fracture and about others he made, Perrin used the terms indirect and contrecoup synonymously. If the head, when dropped, was entire, as the account by Perrin implies, the question of a possible tug by the face mass when the head struck and the rôle such a force had in pulling the bones of the face away from those of the cranium is worthy of consideration.

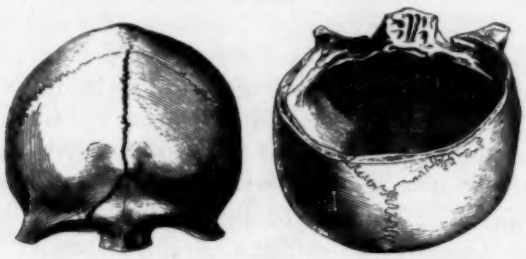

Fig. 10.—An indirect fracture made by Perrin by dropping a head on a cushioned surface so that the occiput struck first. (From Legouest, L.: Traité de chirurgie d'armée, Paris, 1872, p. 212.)

For many years descriptions in textbooks of the dynamics of linear cranial fractures have been based in a large measure on the experiments of Hermann,[49] Schranz [48] and Messerer,[51] and on the bursting and bending theories to which their investigations gave birth.[50] Most of their experiments were made with loose heads, or with the bottom halves of empty skulls. Practical application of the bursting and bending theories has helped to explain involved and apparently disorderly comminution, the "fractures à grand fracas" of Félizet. It has also enabled authors to differentiate the two forms of fractures in illustrations.[109] These descriptions of the way fractures occur, so dominated by conceptions of bursting and bending, fail to mention that the bursting theory was brought forward a decade before the

108. Perrin, M.: Les fractures du crâne par contrecoup, Bull. et mém. Soc. d. chir. de Paris **4**:128, 1878; experiment 4, p. 132.

109. Körber.[60] van Nes.[63]

work of Hermann, by Forgues in his "fracture par éclatement," [28] or that Forgues frankly stated that his explanation was not new; he gave credit for his ideas to Chassaignac,[110] who wrote in 1842:

> Je ne pense pas que l'existence d'une fracture à une certaine distance du point frappé, suppose necessairement une résistance inégale dans les différents points de l'étendue du crâne. La mécanique nous apprend que si l'on soumettait à une forte pression une sphére creuse, élastique, parfaitement homogène de substance et d'épaisseur, la rupture se produirait au sommet des courbes dont la courbure serait augmentée, et non pas dans les points directement soumis à la compression.

There is no clue to the source of Chassaignac's knowledge, nor is it very material whether the meridians start to burst at the equator or at the poles. There is a strong likelihood that Chassaignac's comprehension of bursting fractures was a reflection of the work of physicists with the vibrations of rings and bells.[111] And it is quite probable that the experiments performed by Sir Charles Bell by hitting metal rings with a hammer had a similar origin notwithstanding that they were made in 1816.[27]

The experiments on loose heads by Hermann and others were mainly concerned with cranial architecture and elasticity, and the relations of these to direction of the force. Neither in their experiments nor in the ensuing deductions is the influence of the weight of the trunk or structures of the face given the attention it deserves. Duret,[90] and his pupil Poulain,[74] who used entire bodies, declared that the part of the cranium receiving the blow slips away from the remainder held rigidly to the spine. Baum [112] insisted that the trunk often acts like a one-armed lever with its fixed point where the body is supported by the feet. We believe that the weight of the trunk and its extremities, as well as the inertia of the extracranial part of the head, should invariably be included among other factors responsible for not only the fractures but also the location and other characteristics cranial fractures possess.

SUMMARY

Because they were more or less symmetrical or had split the cranium to some extent in lateral halves, cranial fractures in 80 bodies are described, and explanations are offered for how they occurred. The 80 bodies were among 1,278 with traumatic, linear, cranial fractures disclosed by necropsy.

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110. Chassaignac, E.: Lésions traumatiques du crâne et des parties qu'il contient, Thèse du concour, Paris, 1842.

111. Schröen [10e] mentions the physicists Leibnitz, Wedel and Stosch, but does not give references.

112. Baum, W.: Beitrag zur Lehre von den indirekten Schädelfrakturen, Arch. f. klin. Chir. **19**:381, 1876.

The mechanism of origin and other particulars of ring-shaped fractures in the cranial base in 36 of the 80 bodies are discussed together with the details reported in the literature regarding similar fractures in 45 other craniums.

An attempt is made to account for ring fractures of the base caused by violence at the occiput.

Symmetrical incomplete ring fractures ventrally placed in the vertex and obliquely directed from behind downward and forward observed in 5 craniums are reported, and suggestions are made for the way they were produced.

The usefulness of all relevant evidence in interpreting the mechanism of any traumatic, linear, cranial fracture is illustrated.

Some of the important contributions to the development of modern opinions of the dynamics of traumatic, linear, cranial fractures from blunt force are reviewed.

BILE PERITONITIS

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It has been impossible to correlate laboratory and clinical experience in relation to bile peritonitis because of the striking contradictions in data obtained from the two sources. These contrasting data have not been understood, yet the results of laboratory experiments have been applied to the clinical problem. The results have been incomprehensible, because the two methods of study are incompatible for this subject.

CONTINUOUS EXTRAVASATION OF STERILE BILE

Recently, Wangensteen,[1] Horrall,[2] Rewbridge[3] and others repeated and elaborated on the experiments of earlier workers, and showed that large quantities of sterile bile poured continuously into the peritoneal cavity of an animal will produce death within from six to seventy-two hours. In most of these experiments long incisions or large apertures were cut in the gallbladder or the bile duct, and the abdomen was sutured tightly. Thus Bohn,[4] in 1775, removed the gallbladder in dogs without ligating the cystic duct and found that most of the dogs died. In 1902, Ehrhardt[5] sectioned the choledochus in 12 dogs and cats. In twenty-four hours the animals were slightly jaundiced. Within from two to six days they were intensely jaundiced, and most of them had convulsions; all of them died. In 1910, Noetzel[6] incised the gallbladder of 17 rabbits and closed the abdominal wall. The animals died. In 1921, Fulle[7] found that large incisions in the gallbladders of dogs and rabbits invariably proved fatal. Although other laboratory workers obtained

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From the Department of Surgery, University of California Medical School, and the San Francisco Hospital, Department of Public Health.

1. Wangensteen, O. H.: On the Significance of the Escape of Sterile Bile into the Peritoneal Cavity, Ann. Surg. **84**:691, 1926.

2. Horrall, O. H.: Experimental Bile Peritonitis: Its Treatment in the Dog, Arch. Int. Med. **43**:114 (Jan.) 1929.

3. Rewbridge, A. G.: The Etiologic Rôle of Gas-Forming Bacilli in Experimental Bile Peritonitis, Surg., Gynec. & Obst. **52**:205 (Feb.) 1931.

4. Bohn, quoted by Rost: The Pathological Physiology of Surgical Diseases, Philadelphia, P. Blakiston's Son & Co., 1923.

5. Ehrhardt, O.: Ueber Gallenresorption und Giftigkeit der Galle im Peritoneum, Arch. f. klin. Chir. **64**:314, 1901.

6. Noetzel, W.: Experimentelle Untersuchung zur Gallenblasen Perforations Peritonitis, Arch. f. klin. Chir. **93**:160, 1910.

7. Fulle, G. B. C.: Contributo sperimentale alle ferite della cistifellea e alla colemia sperimentale, Arch. ital. di chir. **4**:229, 1922.

diametrically opposed results, I believe that careful analyses of experimental data prove that death follows when large quantities of bile are continuously extravasated into the peritoneal cavity of the lower animals.

Every constituent of bile has been considered by various experimenters to be the cause of death in these animals, but the work of Wangensteen,[1] Horrall [2] and Ravdin, Morrison and Smyth [8] proves that bile salts are the only factors that can be responsible. However, it has not been proved that they, per se, are the sole cause of death. Numerous experiments have shown that they may be injected intravenously, in relatively large amounts, without sequelae.

Corresponding examples occurring in man are virtually unknown. In many thousands of autopsies, few, if any, comparable illustrations are afforded. Thus Robertson,[9] Rusk,[9] Conner [9] and Ophüls [9] reported that they remembered in their total experience only 1 or 2 cases in which diffuse, sterile bile peritonitis emanating from a constant outpouring of bile caused death. In a series of 377 necropsies in which I found grossly diseased gallbladders, no instances of bile peritonitis were encountered.

Surgical experience has offered a few illustrations of death following slippage of the tie of the cystic duct or severance of anomalous biliary ducts. Extremely few reports have been published relative to the frequency of this catastrophe in surgery. Personal conversations have elicited citations of only one or two instances in the memory of some of my notable confrères. Most of the cases which were recalled did not fall into the category of diffuse sterile bile peritonitis, or death from this source was not proved. When the records of remembered cases were reviewed, I found that the data were usually inadequate for critical study, or that the illustration cited was not one of sterile bile peritonitis.

Clinically also, sterile bile peritonitis seems rare and relatively innocuous.[10] In 1735, Wedekind [11] noted a case of intraperitoneal escape of bile after traumatic wounds of the liver, with recovery of the patient. In 1785, Skeete [12] recorded the case of a boy who fell from a tree and lacerated the gallbladder. Death did not occur until six weeks later, when the peritoneal cavity was found filled with bile-stained fluid. Paracentesis was performed on the twenty-fourth day after injury, when 16 pints (8 liters) of bile-stained transudate was removed, and at

8. Ravdin, I. S.; Morrison, M. E., and Smyth, C. M.: Bile Peritonitis and Bile Ascites, Ann. Surg. **89**:867, 1929.

9. Personal communication to the author.

10. Mentzer, S. H.: A Clinical and Pathological Study of Cholecystitis and Cholelithiasis, Surg., Gynec. & Obst. **42**:782 (June) 1926.

11. Wedekind, A. J.: De vulnere hepatis curato, Jena, lit. Mullerianis, 1735.

12. Skeete, Thomas: Case of Considerable Effusion of Bile into the Cavity of the Abdomen in Consequence of a Fall; with the Appearance on Dissection and Some Additional Remarks, London M. J. **6**:274, 1785.

autopsy, when from 2 to 3 gallons (8 to 11 liters) of similar fluid was encountered. Fryer [13] reported the case of a boy, aged 13, who was struck by a wagon shaft. Two weeks later a mass appeared over the region of the gallbladder. On aspiration, 13 pints (6 liters) of bile-stained fluid was withdrawn. Twelve days later 15 pints (7 liters) was removed; nine days later, 13 pints, and twenty days later, 6 pints (3 liters). The boy recovered without further aid. In 1843, Budd [14] reported the case of a man who was struck in the abdomen by a heavy ladder. During the four months after his injury he was tapped eight times, and bile-stained transudate was removed. In 1845, Abraham Coles stated that in his experience not every puncture of the gallbladder was fatal. In 1882, von Pitha and Billroth [15] pointed out that the liver and gallbladder have frequently been punctured by saber and bayonet wounds, and that the patients have lived. In 1884, Smarth [16] removed a projectile from the liver. Considerable bile had oozed into the peritoneal cavity, yet the patient recovered. Petersen [17] aspirated 5,600 cc. of bile from the peritoneal cavity of a boy traumatically injured. Five weeks after injury two rents in the gallbladder were successfully sutured. In most saber, bayonet and projectile wounds involving the liver or gallbladder, organisms enter the peritoneal cavity. Some of the aforementioned cases were therefore not instances of sterile bile peritonitis, yet death resulted late, if at all.

In 1829, Compaignac [18] described the case of a man, aged 35, who received a violent blow in the abdomen. He did not die until eighteen days later, yet autopsy showed a rent in the left hepatic duct and 6 pints of deep green bile in the peritoneal cavity. In 1891, Treves [19] pointed out that in choledochotomies it was often necessary to insert gauze drains about the incised common duct and at the same time to drain the pouch of Douglas "abdominally or vaginally" with glass tubes to "care for the drainage of bile." Landerer [20] reported a case of rupture of a bile duct in which sixteen successive punctures of the abdominal cavity were

13. Fryer: Case of Extravasation of Bile into the Cavity of the Abdomen from Rupture of the Liver or Gallbladder, Tr. Med.-Chir. Soc., London **4**:330, 1813.

14. Budd, George: Diseases of the Liver, Philadelphia, Blanchard & Lea, 1853.

15. von Pitha and Billroth: Handbuch der allgemeinen und speciellen Chirurgie, Stuttgart, Ferdinand Enke, 1882, vol. 3, p. 190.

16. Smarth, quoted by Pantaloni, P.: Chirurgie du foie et des voies biliaires, Paris, Institut Bibliothèque Scientifique, 1899.

17. Petersen, W.: Beiträge zur Pathologie und Therapie der Gallensteinskrankheit, Beitr. z. klin. Chir. **23**:705, 1899.

18. Compaignac, J. A. J., quoted by Terrier and Auvray.

19. Treves, Frederick: A Manual of Operative Surgery, Philadelphia, Lea Bros. & Co., 1892.

20. Landerer, A.: Verletzung der Gallenwege; Gallenerguss in die Bauchhöhle, Heilung, Deutsche Ztschr. f. Chir. **29**:611, 1889.

performed, with a total removal of 35 liters of bile. Garrett [21] removed 16 quarts (15 liters) of bile by repeated aspirations in a man traumatically injured, and twenty days after injury operation showed a tear in the posterior wall of the common duct. Recovery ensued. Uhde [22] removed 14.5 Kg. of bile by paracentesis twenty-three days after injury. Thirty-seven days later, 9 Kg. of bile was aspirated, and the patient recovered. Thompson [23] removed 4 quarts (4 liters) and later 5 quarts (5 liters) of bile and still later five or six aspirations of several quarts each were performed; and recovery ensued. Garré [24] reported the examination of a patient two months after injury, when a large amount of bile was evacuated from the peritoneal cavity; the patient recovered. Hildebrandt [25] found a large quantity of bile in the peritoneal cavity twenty-three days after injury from a tear in the hepatic duct. Ricketts [26] reported 28 cases of traumatic rupture of the biliary tract in which the patients had been operated on, with a mortality of 22 per cent. Meissner [27] found that in 25 per cent of 12 cases of traumatic rupture of the common duct and in 30 per cent of 7 cases of rupture of the hepatic duct recovery occurred. Courvoisier [28] collected 34 cases of rupture of the biliary tract, with 12 recoveries after surgical intervention. Amante [29] collected 101 cases of traumatic rupture of the biliary passages. He believed that without surgical intervention death is sooner or later inevitable. McWilliams [30] stated, however, that normal bile, even in large amounts, is fairly well tolerated by the peritoneum. Many of the aforementioned cases confirm McWilliams' belief, although it is

21. Garrett, R. W.: Traumatic Rupture of the Bile Duct, Ann. Surg. **31**:227, 1900.

22. Uhde, C. W.: Zerreissung eines Gallenganges mit glücklichem Ausgange, Arch. f. klin. Chir. **25**:485, 1880.

23. Thompson, J. F.: Bile in the Abdominal Cavity, Boston M. & S. J. **145**: 186, 1901.

24. Garré, C.: Traumatische Hepaticusruptur geheilt durch eine Hepato-Cholangio-Enterostomie, Beitr. z. Physiol. u. Path., Festschr. L. Hermann, 1908, p. 72; quoted by Thöle.[47]

25. Hildebrandt, W.: Ueber die traumatische Ruptur des Ductus hepaticus, Arch. f. klin. Chir. **81**:646, 1906.

26. Ricketts, B. M.: Rupture of the Gall-Bladder (Spontaneous and Traumatic; With and Without Operation); An Historical Review of 273 Cases, St. Louis M. Rev. **51**:108, 233, 276, 456 and 497; **52**:4 and 25, 1905.

27. Meissner: Die Zerreissungen der Gallenausführungs-Gänge durch stumpfe Gewalt, Beitr. z. klin. Chir. **54**:204, 1907.

28. Courvoisier, L. G.: Casuistich-statistiche Beiträge zur Pathologie und Chirurgie der Gallenwege, Leipzig, F. C. W. Vogel, 1890, p. 152.

29. Amante, quoted by Kehr.[33]

30. McWilliams, C. A.: Acute Spontaneous Perforation of the Biliary System into the Free Peritoneal Cavity, Ann. Surg. **55**:235, 1912.

evident that surgical drainage is indicated. Kulenkampff,[31] Waugh,[32] Kehr,[33] Barling,[34] Garré,[24] Dirk,[35] Hildebrandt,[25] Fryer,[13] Barlow [36] and others have had cases of recovery after long periods of time. Although in most of the aforementioned cases large wounds were present in the gallbladder or biliary ducts, it is noted that pure bile is not described as being present in the peritoneal cavity in any instance. Adequate descriptions are not found in many of the clinical reports, and undoubtedly many of the deaths were due to secondary infection or to systemic lesions. In the carefully described cases bile-stained transudates were present. One wonders why bile alone was not found. The explanation may come from three possible sources: (1) The patient promptly died before the irritated peritoneum could exude serous material; (2) the bile became localized, i. e., encysted, (3) or an external escape for the bile was effected spontaneously or with the help of aspirations. In none of the patients who died was death proved to be due to sterile bile peritonitis. Those who lived ceased having diffuse bile peritonitis promptly, for localization of the bile occurred by adhesion encystment, or surgical drainage was effected. Obviously, then, sterile bile peritonitis has not been proved to be a cause of death in these cases or, indeed, of a serious consequence. It is possible that nature will not permit extravasated sterile bile to remain long enough in man to become serious, for it is changed into a localized form or transudation develops so rapidly that the ascites must be surgically drained. Adequate supportive treatment, of course, must be maintained.

Guibe [37] concluded that sterile bile in the peritoneal cavity is innocuous. McWilliams, Arth, Sick and Fraenkel,[38] Buchanan,[39] Ritter,[40]

31. Kulenkampff, D.: Traumatische Zerreissung der grossen Gallenwege mit Ausgang in Genesung, Zentralbl. f. Chir. **12**:757, 1885.

32. Waugh, G. E.: Traumatic Rupture of the Common Duct in a Boy, Brit. J. Surg. **3**:688, 1916.

33. Kehr, Hans: Chirurgie der Gallenwege, in von Bruns: Neue Deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1913, vol. 8; Ueber einen durch ideale Cholecystotomie geheilten Fall von Schussverletzung der Gallenwege, Zentralbl. f. Chir. **19**:645, 1892.

34. Barling, H. G.: Case Report, Tr. Midland M. Soc., Lancet **1**:34, 1901.

35. Dirk: Traumatische Choledochusruptur, Zentralbl. f. Chir. **33**:783, 1906.

36. Barlow, W. B.: Case of Tumor in Right Hypochondrium Occurring After Injury from Which a Large Quantity of Fluid Resembling Bile Was Repeatedly Withdrawn by the Operation of Tapping, Tr. Med.-Chir. Soc. London **27**:378, 1844.

37. Guibe, M.: Les cholépéritoines avec intégrité apparente des voies biliaires, Rev. de chir., Paris **49**:233, 1914.

38. Sick, C., and Fraenkel, E.: Ein Beitrag zur sogenannten galligen Peritonitis, Beitr. z. klin. Chir. **85**:687, 1913.

39. Buchanan, J. J.: Bile Peritonitis Without Evident Perforation of the Biliary Tract, Surg., Gynec. & Obst. **26**:303, 1918.

40. Ritter, C.: Die gallige Peritonitis ohne Perforation, Arch. f. klin. Chir. **118**:54, 1921.

Burckhardt,[41] Hearn,[42] Terrier and Auvray [43] agreed. Gibbon,[44] and, later, Schlatter,[45] found that sterile bile was innocuous in the peritoneal cavity unless absorbed in large amounts. Courvoisier concluded from studies of animals and from clinical experiments that sterile bile in the peritoneal cavity is innocuous even in relatively large amounts. Hermes [46] came to similar conclusions after an experience with a case of extensive bile peritonitis in man due to traumatic perforation of the common duct. Lewerenz [47] reported the case of a boy, aged 2½ years, who suffered a traumatic rupture of the common duct and recovered after operation. Lewerenz collected 63 more cases from the literature, many with recovery. When death occurred, he attributed it to the absorption of biliary salts. Ritter and Burckhardt insisted that death from cholemia following rupture of the bile ducts has never been observed. Stierlin [48] stated that sterile bile contained within the peritoneal cavity for long periods produced death by fatal toxemia. Malnutrition invariably occurred, however, and studies at autopsy indicated that death was due to metabolic disturbances. It has not been demonstrated that encysted bile is absorbed in man in sufficiently large quantities to cause toxemia. Ricketts reported 273 cases of spontaneous and traumatic perforations of the extrahepatic tree; he found a decidedly better outcome in the cases of trauma (i. e., sterile bile). Thöle [49] also came to the same conclusion (see addenda).

During the last twelve years I have had only one case of diffuse sterile (?) bile peritonitis at the San Francisco Hospital:

A woman, aged 45, had had moderately severe attacks of gallstone colic for one year. Seven days before admission to the hospital she had a severe pain followed by vomiting, with localized tenderness and rigidity over the gallbladder. It was believed that she had moderately acute cholecystitis, but as the temperature was normal and the white cell count 9,250, she was treated conservatively. Observation continued for five days, during which time the four hourly temperature readings remained normal and the white count stayed below 9,000. The patient was not

41. Burckhardt, H.: Perforationslose Gallenperitonitis, Beitr. z. klin. Chir. **128**:209, 1932.

42. Hearn, W. J., in discussion on Jopson: Rupture of the Gallbladder with Profound Toxemia, Ann. Surg. **40**:424, 1904.

43. Terrier, F., and Auvray, M.: Les traumatismes du foie et des voies biliaires, Rev. de chir., Paris **17**:16, 1897.

44. Gibbon, J. H., in discussion of paper by Jopson.[42]

45. Schlatter, C.: Die Behandlung der traumatischen Leberverletzungen, Beitr. z. klin. Chir. **15**:531, 1896.

46. Hermes: Case Report, Ann. Surg. **16**:393, 1892.

47. Lewerenz: Ueber die subcutanen Ruptur der Gallenwege traumatischen Ursprungs, nebst einem casuistischen Beitrag, Arch. f. klin. Chir. **71**:111, 1903.

48. Stierlin, R.: Traumatische subcutane Ruptur des Ductus choledochus, Deutsche Ztschr. f. Chir. **73**:462, 1904.

49. Thöle, F.: Die Verletzungen der Leber und der Gallenwege, in von Bruns: Neue deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1912, vol. 4, p. 144.

jaundiced, but because of persistent abdominal tenderness and rigidity, exploration was undertaken. Large pools of bile were found in the peritoneal cavity. Unfortunately cultures were not taken. The gallbladder was perforated and empty except for two mulberry stones, which were removed. A cholecystostomy was performed, and the patient had an uneventful convalescence.

SINGLE LARGE EXTRAVASATION OF BILE

Many authors have showed that large quantities of sterile bile may be poured experimentally into the peritoneal cavity at one time without the occurrence of death. Thus, Malpighi [50] in 1666, made a biliary fistula in a dog and noted that large quantities of bile might be spilled without serious consequences. In 1839, Thomson [51] stated, "In experiments on animals it has been proved that rupture of the gallbladder does not necessarily cause death. These experiments seem to warrant the conclusion that it is not so much the original escape of fluid that causes death as it is the continual seepage of bile into the peritoneal cavity." In 1767, Herlin [52] incised the gallbladders of cats after ligating the cystic duct, thus allowing the bile from the gallbladder to empty into the peritoneal cavity. He then sewed the gallbladder wound together, and the animals lived. At autopsy, several weeks later, no trace of bile was found in the peritoneal cavity, and the gallbladder was filled with a colorless fluid. L'Anglais repeated the experiment on 2 dogs, but he also removed the gallbladder. The dogs lived. Fulle found that small incisions left open in the gallbladder or bile ducts did not prove fatal. Later, examinations at autopsy showed that these small wounds had closed. Noetzel concluded from experiments on dogs that the contents of the gallbladder, when allowed to drain into the peritoneal cavity, produce no untoward results. Bostroem,[53] Emmert,[54] Hering,[54] Villaderbo,[54] Compaignac,[54] Ammusat [54] and Schwartz [54] performed similar experiments with similar results. Wangensteen [1] and Horrall [2] recently repeated the experiments, showing that the sterile contents of the gallbladder may be emptied into the peritoneal cavity with impunity. The peritoneum remains smooth and glistening.

Clinically, single large extravasations of sterile bile into the peritoneal cavity are extremely rare. It is only following trauma that sterile bile is evacuated from the gallbladder into the peritoneal cavity. But even in these cases a constant stream of bile may be poured into the peritoneal cavity through the aperture in the gallbladder wall via the cystic duct.

50. Malpighi, quoted by Budd.[14]

51. Thomson, William: Diseases of the Liver and Bile Passages, New York, E. Barrington & George D. Haswell, 1842.

52. Herlin, quoted by Thomson.[51]

53. Bostroem, quoted by Lesser, in discussion of Thiersch: Zerreissung eines Gallenganges mit todlichem Ausgannge, Verhandl. d. deutsch. Gesellsch. f. Chir. 8:117, 1897.

54. Quoted by Terrier and Auvray.[45]

It is extremely difficult to conceive of a pathologic condition in which, although the cystic duct is obstructed and the gallbladder wall and the gallbladder bile are sterile, a perforation of the gallbladder nevertheless occurs. Theoretically it is possible, but I have not been able to find a record of it. When clear bile is emptied from the gallbladder following an acute or subacute spontaneous perforation, the bile cannot be considered sterile, even though one or two swabs are taken from the peritoneum for culture. Perforations of this nature presuppose infection of the gallbladder wall, and when the wall is ruptured organisms are presumably scattered into the adjacent peritoneal areas. It is difficult to obtain cultures of organisms from single samples of bile from the gallbladder. Frequently only one of several swabs will yield a positive culture, even under these ideal circumstances. It is therefore likely that one or two samples taken from the peritoneum will yield no growth. Under these circumstances one is not justified in concluding that the extravasated bile is sterile. Much less may one overlook the site of infection at the perforation in the gallbladder wall. It is a common experience in the operating room, however, to note the spilling of small quantities of gallbladder or hepatic bile. Even when large quantities of bile escape during operations on the gallbladder, postoperative complications are rarely encountered. This may be due to a fear inherent in the surgeon's mind that leakage of bile will occur, for such areas are usually drained. However, in the vast majority of patients, extravasated bile is rapidly walled off by adhesions. When spilled bile is infected, drainage should be provided, but I have occasionally closed the abdomen tight without ill consequences even in cases of this sort.

INJECTIONS OF LARGE QUANTITIES OF BILE

Conflicting results have been obtained in the laboratory when large quantities of sterile bile were injected experimentally through the abdominal wall. Fraenkel and Krause [55] injected bile in varying quantities into the peritoneal cavity of guinea-pigs and dogs and found that it was well tolerated. Fulle noted that small quantities of bile from the gallbladder, when injected intraperitoneally, caused no reaction, but that large amounts proved fatal to dogs and rabbits. McWilliams found that large quantities injected into the abdominal cavity caused little trouble. Wangensteen made injections of sterile bile from the gallbladder into 3 rabbits. All of them died within five days, and all except 1 of 12 guinea-pigs died within twelve hours. The chief symptoms were lethargy, anorexia, diarrhea, rapid respiration and jaundice. At autopsy the peritoneal cavity was normal except for areas of necrosis in the liver where bile came into contact with Glisson's capsule. The kidneys showed acute nephritis, and the cortical cells of the suprarenal

55. Fraenkel, E., and Krause, P.: Bakteriologisches und Experimentelles über die Galle, Ztschr. f. Hyg. u. Infektionskr. **32**:97, 1899.

glands were markedly degenerated. Beyond a certain quantity, additional injections of bile caused no increased reaction or rapidity of death, nor did the reactions caused by bile from the liver and gallbladder differ. Horrall claimed that when large quantities of sterile bile were continuously emptied into the peritoneal cavity of animals death ensued from a toxemia produced by the absorption of bile salts. Rewbridge repeated Wangensteen's and Horrall's experiments, but found that the peritoneum, instead of remaining grossly normal in appearance, was fiery red and covered with fibrinous exudate. Moreover, he constantly obtained positive cultures of Bacillus Welchii. He was not able to find sufficient bile salts in the blood to cause death. Even when large quantities of bile salts were introduced into the peritoneal cavity, no significant rise in the bile salts of the blood was found.

Thus the laboratory has furnished three groups of conflicting evidence: 1. Many workers have found that large quantities of sterile bile drained or injected into the peritoneal cavity were entirely innocuous. Bacteriologic cultures were uniformly negative. 2. Others observed death within from six to one hundred and twenty hours after similar experiments without evidence of peritoneal reaction or infection, but with marked changes in the kidneys and suprarenal glands. They held that bile salts are absorbed and are toxic. 3. Rewbridge found, after similar experiments, an extensive inflammation of the peritoneum with positive cultures of anaerobes (B. Welchii) almost as a routine. He concluded, as did Dorsey,[56] in 1818, and Thomson,[51] in 1839, that bile salts react on the intestinal walls, making them permeable and thus permitting the escape of bacteria from the intestinal lumen. In 1931, Meleney, Harvey and Jern [57] expressed the opinion that after a seepage of bile had occurred, peritonitis developed secondarily from organisms which had permeated the intestine.

These three groups of conflicting results from laboratory workers seem difficult to correlate. Some assistance, however, is afforded by other experimental evidence. In 1797, Eaglesfield Smith [58] immersed flesh in bile for fourteen hours at body temperature and found that it lost half its weight. From this evidence he concluded that bile was a digestant of tissue. In 1866, Leyden [58] noted that bile acids caused a degeneration of the musculature of the blood vessel walls. Several observations have been made of edema and even of necrosis of the muscles of the abdominal wall in patients with extensive bile peritonitis.

56. Dorsey, J. S.: Elements of Surgery, Philadelphia, E. and R. Parker and B. Warner, 1818, vol. 1; quoted by Budd.[14]

57. Meleney, F. L.; Harvey, H. D., and Jern, H. Z.: Peritonitis: I. Correlation of the Bacteriology of the Peritoneal Exudate and the Clinical Course of the Disease in One Hundred and Six Cases of Peritonitis, Arch. Surg. **22**:1 (Jan.) 1931.

58. Quoted by Legg, J. W.: Bile, Jaundice and Bilious Diseases, New York, D. Appleton & Company, 1880.

In 1874, Fleischle [58] tied the common duct of dogs and connected the liver end to the thoracic duct by means of a cannula. Though an abundance of bile acids were recovered from the lymph ducts, none was obtained from the blood stream, indicating that they were destroyed by the blood or were too greatly diluted to be recoverable. In 1931, Rewbridge was likewise unable to detect increased amounts of bile acids in the circulating blood of animals dying from extensive bile peritonitis. He therefore concluded that circulating bile acids could not be the cause of the toxemia.

If, then, extensive sterile bile peritonitis is so rare in man, how can one correlate the findings of the laboratory relative to its seriousness? Should one not expect the condition in man to be present frequently and to be the cause of death? Probably the laboratory itself has answered these questions, for Brand [59] showed that dog's bile contains eight times as much taurocholic acid as glycocholic acid, while human bile contains only from one-fifth to one-eighth as much. Furthermore, taurocholic acid is from twelve to twenty times as toxic as glycocholic acid. Therefore bile peritonitis in a dog is at least forty times as dangerous as a proportionate amount of bile peritonitis in man. In rabbits and guinea-pigs a similar, though less marked, disparity is found, yet from such experiments conclusions are drawn and appropriated to man. I believe that the laboratory animal itself is responsible for many of the divergent opinions which have arisen relative to experimental bile peritonitis. Meyer,[60] director of the Hooper Foundation, informed me that the livers of most dogs, rabbits and guinea-pigs are constantly filled with organisms. Animals used for experiments on bile peritonitis are frequently not satisfactory, even when raised and fed under strictly aseptic conditions. The liver is a virtual incubator for organisms, and it is common experience to culture aerobes, as well as anaerobes, from the bile of animals which have arrived at the experimental laboratory through the usual channels.

ENCYSTED BILE

In 1823, Cooper [61] quoted a case of Calacin's in which a large extravasation of bile was limited by adhesions, and the patient recovered. Labrosse [62] reported a case of a boy aged 17, who fell from a ladder onto a wall, striking the right hypochondrium. Eight or ten days later the patient's abdomen began to swell and continued protruberant for two years, when Jaboulay operated on him. Ten liters of bile was found

59. Brand, J.: Beitrag zur Kenntnis der menschlichen Galle, Arch. f. d. ges. Physiol. **90**:491, 1902.

60. Meyer, Karl: Personal communication to the author.

61. Cooper, Samuel: A Dictionary of Practical Surgery, New York, Collins & Hannay, 1823.

62. Labrosse: Contribution à la étude du cholépéritoine spontané, Thèse de Lyon, no. 86, 1912.

encysted about the gallbladder, which had a small patent opening that had evidently persisted for two years. In 1925, Polya [63] reported a case in which jaundice, clay-colored stools, an enlarged liver and fever developed seven days after cholecystectomy. Eight months later the patient was operated on again, and a cyst containing at least 200 cc. of pure bile was found at the operative site. Similar cases of prolonged encystment of bile were reported by Drysdale,[64] Thiersch [65] and Rätjen.[66] Certainly it is known that bile may remain encysted for long periods without serious sequelae.[67] Enderlin and Justin [68] showed that lacerations in the gallbladder wall close rapidly. Therefore diffuse extravasations of bile are prevented. Furthermore, the pressure of bile secretion is low, and this fact, coupled with the readiness of visceral and omental adhesions, permits the rapid closure of wounds in the gallbladder wall and the ready development of encystment. Recently I drained 600 cc. of infected bile which had localized about the subhepatic area when a T-tube leaked following choledochostomy. The accumulation of infected bile appeared as a mass at the operative site six days postoperatively without other clinical evidence of its development, and in spite of adequate drainage through the T-tube. Two Penrose drains leading to the site of the operation also failed to drain away the bile thus accumulated. For seven weeks postoperatively a large pool of bile (approximately 450 cc.) remained localized in this area without serious sequelae. When it was drained by a third operation, convalescence was uneventful.

INFECTED BILE

Infected bile which has spread diffusely through the peritoneal cavity is recognized by most authorities as being extremely dangerous. Although bile is considered by most bacteriologists to be an inhibitor of bacterial growth, bile and pus, when emptied into the peritoneal cavity of man, seem to increase the virulence of pyogenic peritonitis. Numerous cases of acute ulcerative perforations of the gallbladder and bile ducts in which large quantities of infected bile were widely dis-

63. Polya, E.: Gallencyste nach idealer Cholecystektomie, Zentralbl. f. Chir. **52**:2341, 1925.

64. Drysdale, T. M.: Case of Rupture of the Common Duct of Liver: Formation of Cyst Containing Bile. Death Occurring on Fifty-Third Day, Am. J. M. Sc. **41**:399, 1861.

65. Thiersch, K.: Zerreissung eines Gallenganges mit tödlichem Ausgange, Verhandl. d. deutsch. Gessellsch. f. Chir. **8**:117, 1879.

66. Rätjen: Ueber einen Fall von traumatischer Gallencyste, Deutsche med. Wchnschr. **13**:155, 1887.

67. Mentzer, S. H.: Acute Cholecystitis: Its Surgical Treatment, California & West. Med. **32**:224 (April) 1930.

68. Enderlin and Justin: Ueber die Heilung von Wunden der Gallenblase und die Deckung von Defecten der Gallenblase durch transplantiertes Netz, Deutsche Ztschr. f. Chir. **61**:235, 1901.

seminated throughout the peritoneal cavity have resulted in mortalities of from 48 to 100 per cent, depending on whether or not operative aid was immediate.[69] The abundant literature relative to acute cholecystic perforations during typhoid fever and parasitic infections attests to almost uniform fatality in infected, diffuse bile peritonitis unless surgical aid intervenes.

In 1757, Boerhaave [70] recorded the case of a man whose gallbladder was run through by a sword, and who died in three days, "convulsed." Watson [71] reported the case of a man whose gallbladder was perforated by a bayonet wound and who died within thirty-six hours. In 1812, Sabatier [72] expressed the opinion that extravasated, infected bile is absolutely fatal. In 1818, Dorsey concluded that wounds of the gallbladder are fatal because the extravasated bile irritates or disturbs the function of the peritoneum. In 1839, Thomson pointed out that perforation of the gallbladder, with infected bile, is "absolutely fatal" unless operation is performed.

In 1903, Erdmann [73] reported 33 instances of spontaneous rupture of the gallbladder occurring in typhoid fever. Twenty-seven of the patients were not operated on, and all of these died. Of 7 who were operated on, 4 recovered. During the past twenty years at the San Francisco Hospital only 1 case of perforation of the gallbladder has occurred in association with typhoid fever:

A Filipino boy, aged 20, was admitted to the hospital with a diagnosis of typhoid fever. His temperature ranged from 101 to 105 F. during a three week period, and he was critically ill. There followed a quiescent period of two weeks, during which the temperature ranged from 99 to 100 F. Then, over a four week period, a series of attacks characterized by chill, sudden rise in temperature and absence of pain began. Finally a severe pain about the unbilicus, with high temperature, rapid pulse and severe chill, developed. An acute condition of the abdomen was recognized and the patient was operated on within twenty-four hours. A perforated gallbladder with extensive bile peritonitis, yielding a culture of Bacillus typhosus, was encountered. One gallstone was found and removed, and the gallbladder was drained. The patient had an uneventful convalescence. The drain was removed on the fourth day, and no bile exuded from the wound thereafter. The contents of the gallbladder obviously emptied into the peritoneal cavity, and no further spilling occurred.

In 1890, Courvoisier [28] collected 34 instances of rupture of the biliary passages following trauma in which 22 of the patients died and 12

69. Mentzer, S. H.: The Acute Gallbladder Manifesting Few Signs or Symptoms, Surg., Gynec. & Obst. **55**:716 (Dec.) 1932.

70. Boerhaave, H.: Causus et signis acutorum, et diuturnorum morborum, Lugduni Batavorum, J. Vander, 1735.

71. Watson, quoted by Thomson.[51]

72. Sabatier, quoted by Cooper.[61]

73. Erdmann, J. F.: Primary Typhoidal Perforation of the Gallbladder, Ann. Surg. **37**:878, 1903.

recovered. None recovered without operation or puncture. Only 5 patients died within twenty-four hours, and the sixth died within forty-eight hours. Hemorrhage from the liver was present in 3 cases. In 1905, Ricketts recorded 273 cases of spontaneous or traumatic ruptures of the biliary tract, 224 of which were instances of perforation of the gallbladder alone. Sixty-four patients were operated on; 43 recovered, and 21 died. One hundred and sixty were not operated on; 6 recovered, and 154 died. Therefore, 67 per cent recovered following surgical treatment, while only 3.7 per cent recovered without surgical treatment. Of the 6 patients who recovered without operation none apparently had intraperitoneal drainage of bile. In Ricketts' 49 cases of traumatic rupture of the gallbladder, 29 of the patients were operated on; of these 22 recovered, and 7 died. Of the 20 patients who were not operated on, only 4 recovered,, while 16 died. The cause of death as demonstrated by necropsy was purulent peritonitis. In 2 of the 4 patients who recovered without operation, the establishment of a fistula prevented death. In the third no evidence of intraperitoneal drainage was present. In 1912, McWilliams reported 108 cases of spontaneous perforation of infected biliary tracts. Even though these patients received surgical treatment, the mortality was 48 per cent. In 1918, Buchanan [39] collected 17 cases of rupture of the gallbladder with a mortality of 9 of the 17. Twelve of the 14 patients whose cases were complicated by infection were treated surgically, with a mortality of 50 per cent. The patients who were not operated on died. In 8 cases the duration of symptoms varied from one to six days, with a mortality of 50 per cent, and in 5 cases the duration varied from seven to twenty-seven days, with a mortality of 80 per cent.

During the past twelve years 24 cases of perforated gallbladder have occurred at the San Francisco Hospital. In 8 cases infected bile spread into the peritoneal cavity (table). In 16 cases pus alone exuded. In no instance was sterile bile found in the peritoneal cavity. The 4 patients who were not operated on died. Six of the 20 who were operated on died. One of these lived for fourteen days, until extensive pyogenic peritonitis proved lethal. Three patients died within three days from pyogenic peritonitis which was already present at the time of operation. Two died from shock within twelve hours after surgical intervention.

The careful studies of Billings and Walkling [74] show that most patients who have received stab or gunshot wounds of the biliary system die of hemorrhage or pyogenic peritonitis. Bile peritonitis rarely arises from injuries of this sort. Of 13 patients who had received stab wounds of the liver, 10 were operated on, and hemorrhage was found in all. Death occurred in 7 cases. In only 1 instance was bile extravasated.

74. Billings, A. E., and Walkling, A.: Penetrating Wounds of the Abdomen, Ann. Surg. **94**:1018 (Dec.) 1931.

Death occurred in this case from pneumonia. One patient had a stab wound of the gallbladder. Hemorrhage and bile exuded, but the patient recovered after surgical intervention. Forty-two instances of gunshot wounds of the liver occurred. All of the patients had hemorrhage at operation, and 20 died. Two also had bile extravasations; 1 of these recovered, and the other died of hemorrhage. One patient with a gunshot wound of the gallbladder was operated on but died of hemorrhage. One died of hemorrhage before an operation could be performed. No operation was performed in 5 cases of gunshot wound of the liver, and all of the patients died of hemorrhage.

Data on Eight Patients with a Perforated Gallbladder

Hospital Number	Days in Hospital	Temperature, F.	Blood Count*	Pathologic Data	Operation and Results
D 96898	5	98.6 for 5 days	9,250 86	Large pools of bile free in peritoneal cavity; no pus, but two stones	Cholecystostomy; recovered
D 28549	12	96.6-100	16,000-18,000 89	Gangrenous gallbladder with numerous perforations; 500 cc. of bile-stained pus; stone the size of pigeon's egg in common duct	Cholecystectomy; T-tube in common duct; recovered
D 50434	Few hours	99	17,000 92	Perforated gangrenous gallbladder with large collection of walled-off bile; one stone in common duct	Cholecystectomy; died on 14th day of peritonitis
D 56929	19	99.8-104	12,000 83	Perforation with clear bile diffuse in peritoneal cavity, purulent drainage following; no stones	Drainage of area only; recovered
D 68430	1	98.6	Not done	Perforated with bile in peritoneal cavity	Drainage of area only; died of shock on same day
D 18080	2	99-102	18,400 88	Acute friable hydrops; bile-stained peritoneum; one stone the size of golf ball	Cholecystostomy; recovered
D 40509	10 hours	98.8	11,200 80	Gangrene and perforation, bile in peritoneal cavity; no stones	Cholecystostomy; died same day
D 28504	12	98	18,000 89	Several perforations; bile and pus in peritoneal cavity; stones in gallbladder and common duct	Cholecystectomy and choledochostomy; recovered

* White blood cells and polymorphonuclears (per cent).

The conclusion is inevitably drawn that infected bile which has spread into the peritoneal cavity is extremely serious. Most of the aforementioned cases were instances of peritonitis arising from perforations from which frank pus exuded. Only a few were instances of discharge of purulent bile. The patients obviously died of pyogenic peritonitis. The possibility of pyogenic peritonitis arising in man from digestion of the intestinal musculature by extravasated sterile bile cannot be seriously entertained. I have not been able to find a case which might be used as an illustration of this thesis. Furthermore, the marked contrast in the bile salt content of human and animal bile and the absence of anaerobic cultures in man when bile peritonitis is present must be remembered.

When bile peritonitis is suspected to be the cause of death in man, careful examinations have usually revealed pyogenic peritonitis rather than bile peritonitis. The peritoneum may be stained with bile, but the latter is only a contaminant. In many cases of suspected bile peritonitis the patients have died of hemorrhage, as Billings and Walkling [74] showed. And finally, the extreme rarity in man of diffuse, sterile bile peritonitis must cast doubt on its actual occurrence, for the possibilities of its presence are generously abundant.

I have been unable to find on record in the department for pathology of the San Francisco Hospital a case in which sterile, diffuse bile peritonitis was the cause of death.

CONCLUSIONS

1. Experiments show that bile peritonitis in animals is not comparable to that in man.

2. Diffuse, sterile bile peritonitis has rarely, if ever, been found as the cause of death in man.

3. Extravasated sterile bile is rapidly encysted in man and then becomes relatively innocuous.

4. If the bile is not encysted it provokes ascites, which soon invites surgical intervention.

5. Infected bile which has spread diffusely over the peritoneal cavity proves fatal unless promptly drained by surgical measures. Death ensues from pyogenic rather than chemical peritonitis.

ADDENDA

Since writing this article I have seen an excellent illustration of my thesis at an autopsy performed by Dr. D. A. Wood.

A man, aged 61, who had been drinking heavily, fell, striking the abdomen on the edge of a table. He vomited persistently for one day and then became very ill. He entered the hospital twenty hours later. The temperature was 99.4 F., and the white blood cells numbered 15,400. The abdomen was distended, rigid and tender, particularly to the right of and above the umbilicus. The patient refused to undergo an exploratory operation and died six days after the trauma.

At autopsy, 2,000 cc. of thin, syrupy, yellowish-brown fluid resembling bile was found in the peritoneal cavity. The peritoneum had a greenish tint, and the loops of jejunum were moderately distended and had a slight deposition of fibrin in a few places. The gallbladder had a large tear in its proximal third, from which normal bile was recovered. There was no evidence of gross cholecystic disease.

Advanced bilateral bronchopneumonia accounted for the patient's death. Microscopic examination showed that the kidneys and the suprarenal glands were essentially normal. The liver showed moderately advanced cirrhosis, and the wall of the gallbladder was essentially normal. Bacteriologic examination of the peritoneum showed nonhemolytic streptococci, Staphylococcus albus and Bacillus coli.

I. THERAPEUTIC USE OF BACTERIOPHAGES AGAINST THE COLON BACILLUS

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The preparation and use of bacteriophages against the colon bacilli present special problems. The difficulties appear to be due in part to the numerous varieties and strains of bacteria included within this group and in part to the long continued association between the colon bacilli and various lytic agents of the bacteriophage group in the intestines of man and other vertebrates. A great variety of possibilities is thus presented.

Furthermore, the location of the pathologic condition in the patient may introduce additional complicating circumstances. The infectious process may be located on a surface in contact with abundant other bacteria, as in intestinal ulcer, or in contact with a more or less irritating or toxic liquid, such as the urine, in a wound or in the interior of the blood vessels. The special conditions present at the site of the infection may require careful consideration and sometimes modification in order to permit success. These regional factors are, however, of secondary importance. The primary problem is that of combat against a specific bacterial parasite which is causing the infectious disease. The specific bacteriologic diagnosis is paramount, a point sometimes neglected in the modern enthusiasm for regional specialization in medicine and surgery.

Continued study of bacteriophages discloses the complexity of this group of entities and differences in character of the various lytic agents. Those which act against the colon bacilli present peculiar problems. In preparing for laboratory service in this field one should first collect a large number of bacteriophage races from various sources. These may be obtained from samples of sewage as well as from individual samples of intestinal contents. It is also easy to get them by transfer from other laboratories which may already possess multiple races in their collections. Probably many of these races are not pure but represent mixtures of

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lytic agents associated together. These bacteriophage races are tested against various culture strains of bacteria, and when lysis of the bacteria takes place one carries out serial filtration and inoculation of the filtrate into cultures of the particular susceptible bacterial strain in order to enhance the activity of the bacteriophage to maximum potency. Then this filtrate of enhanced potency is tested against many other bacterial strains to gain an idea of its polyvalence. In practice, one then combines several of the most potent and most polyvalent filtrates for use as a stock polyvalent colon bacteriophage. The individual bacteriophage races are, however, preserved. They keep well in sealed ampules in the refrigerator. In general, the procedure of serial filtration and inoculation of successive cultures results in increased potency of the bacteriophage not only for the individual bacterial strain employed but for other strains. There are, however, exceptions to this. Sometimes the potency diminishes and eventually disappears in serial filtration experiments, and sometimes as the potency increases for one bacterial strain it becomes diminished for another. The assumption that the original bacteriophage actually represents a mixture finds support in such experiences, but one can hardly pretend that the matter is fully understood.

The idea of therapeutic use of the colon bacteriophage comes into consideration when it is recognized that the patient has ulceration of the intestinal mucous membrane, where the presence of colon bacilli may be presumed, or when cultures of the body fluids, such as the urine or blood, or cultures of wound exudate reveal the presence of colon bacilli in these specimens. The first step in the laboratory is the isolation and recognition of the bacterial parasite. Only under conditions of exceptional urgency is one justified in employing a mixed stock colon bacteriophage in treatment before there has been time for specific testing of the bacteriophage against the specific strain in the laboratory. Fortunately, in most instances the condition of the patient does not require immediate emergency use of bacteriophage in infections with organisms of this group. One therefore isolates the bacterial organism in pure culture, tests its various cultural and serologic characteristics and at the same time subjects it to serial filtration studies, at first with the mixed potent colon bacteriophage and subsequently, if necessary, with several of the individual potent races of bacteriophage, in the hope of obtaining complete lysis and, by filtration of such lysed cultures, an autogenous strain-specific bacteriophage of high potency. In desperate cases this work is pushed by continuous twenty-four hour service in the laboratory, especially in those urgent cases in which the treatment has been initiated at once with the stock bacteriophage.

Bacteriophage therapy requires not only skilful, diligent and prompt action in the laboratory but a certain understanding on the part of the physician at the bedside of the behavior of this group of agents and

the conditions for their successful use. Possession of a keen scalpel is not the only requirement for success as a surgeon. Yet one sometimes finds the physician or surgeon fully satisfied with his preparation for bacteriophage treatment as soon as he has read some commercial advertising and has obtained a supply of filtrate from the laboratory. This not only leads to unfortunate results for the patient but tends to discredit bacteriophage therapy in general. The methods of clinical application are still in the process of being perfected, but certain principles are well established. These should not be lightly regarded. Furthermore, the progress of the case needs to be checked by frequent bacteriologic study of specimens from the infected region. Intimate and sympathetic cooperation between the clinician and the bacteriologist is therefore of great importance.

Effective action of the bacteriophage on the microbes cannot be expected unless it comes into direct relation with them. When the infected region is easily reached without risk of damage to the tissues, it is best to place the bacteriophage in or on the lesion. When the infected site cannot be directly approached with safety, intravenous use of the bacteriophage, so that this agent may reach the infected tissues through the blood stream, should be carefully considered. The probable difficulties and dangers of different methods of application should be thoughtfully weighed in the light of experience in the use of bacteriophages. Intravenous bacteriophage therapy requires a special sort of bacteriophage preparation. In our experience a sterile filtrate of a lysed culture grown in protein-free asparagine medium is the most satisfactory type of preparation for this purpose.

When one has to deal with an infection of the urinary tract, in which the colon bacilli are present in the urine within the lumens of the bladder, ureter and renal pelvis and have penetrated into the lymph spaces beneath the epithelial lining of these structures, particularly that of the renal pelvis, and have ascended more or less along the collecting ducts into the medulla or even into the cortical substance of the kidney or may even have established themselves in the blood stream, the problem is not simple. The most dangerous inflammatory lesions in such a case can be reached only through the blood stream, and it is best to begin the use of the bacteriophage by intravenous administration. However, when the infection of the renal tissues has been overcome, one will find new difficulties confronting one in attempting to eradicate the bacteria from the urine in the lumens of the renal pelvis, ureter and bladder. If the urine is markedly acid or alkaline, the colon bacilli in it are able to resist even the most potent bacteriophage. This has been clearly demonstrated by experiments in the test tube in which human urine adjusted to various degrees of acidity and alkalinity from p_H 5 to 8 was employed as the

culture medium.[1] Only in those tubes in the intermediate *p*H range, from about 6.6 to 7.2, was the bacteriophage able to exhibit its full lytic power. Obviously, therefore, one cannot expect bacteriophage lysis to occur in urine within the body if the chemical reaction of this medium is far from the neutral point. Fortunately, the importance of regulating the hydrogen ion concentration of the urine in pyelitis had been recognized as a result of clinical experience before bacteriophage therapy came into the field. In fact, there is good reason to believe that the usual defensive agents of the body, such as the white blood cells and the antibacterial substances of the blood plasma and the lymph, find themselves seriously at a disadvantage in a medium which is very acid or very alkaline. The urine may be made almost neutral in reaction and may be maintained in this condition by the ingestion of a fairly uniform diet, large amounts of fluid and the precisely necessary amounts of alkali-residue drugs, such as potassium citrate or the more powerful and more dangerous sodium bicarbonate, or of acid-residue drugs, such as acid phosphates or ammonium chloride. Determination of the hydrogen ion concentration of the freshly passed urine, each morning and evening, by use of phenol red, bromcresol purple and suitable comparators is the necessary guide to these adjustments, which, moreover, should be made gradually.

As a general rule, chemical antiseptics should not be used in conjunction with bacteriophages, as the lytic agent is inhibited by very minute quantities of such agents as formaldehyde, in fact by quantities too minute to exert evident effect on the bacteria.[1]

It has also been shown that the admixture of blood, blood serum or purulent exudate [2] with urine tends to interfere with the action of the bacteriophage, and it appears that mucus may act in a similar way. Local trauma to the urinary tract by instrumentation may therefore be expected to favor the bacteria as against the bacteriophage. This should receive due consideration before urologic instrumentation is undertaken.

Another matter to be thought about before introducing instruments into the ureters or renal pelves is the danger of spreading the infection to previously uninvolved locations. It is an honored surgical principle that one should not incise through an infected region into a region free from infection. For example, the experienced abdominal surgeon does not open the peritoneal cavity by an incision passing through a furuncle of the abdominal wall. When, therefore, one is certain that the bladder urine is infected with colon bacilli, it is well to pause for consideration before passing an instrument through this bacterial culture into a possibly

1. Frisbee, Frances C., and MacNeal, Ward J.: The Behavior of Escherichia Coli and Its Specific Bacteriophage in Urine, J. Infect. Dis. **46**:405 (May) 1930.

2. Applebaum, Martha, and MacNeal, Ward J.: The Influence of Blood and of Exudate on the Action of Bacteriophage Against the Colon Bacillus, J. Infect. Dis. **50**:269 (March) 1932.

bacteria-free region beyond. This surgical principle should be honored by the observance rather than by the breach, as sometimes obtains in the urologic specialty.

These suggestions are not intended as reflections on the technical procedures of modern urologic instrumentation when properly employed, with adequate knowledge of the bacteriology of the urinary tract, but they are intended as a protest against ill-considered instrumental meddling with the urinary tract during the attempted application of bacteriophage therapy to this region.

Although the available evidence from autopsies and from animal experimentation is rather meager, we are nevertheless convinced that successful bacteriophage therapy of urinary infections proceeds from above downward. The bacteria are defeated and driven out of the renal substance, the renal pelvis and the ureter in turn. In the urinary bladder they are less vulnerable, possibly because of the larger volume of urine, which may have undergone undetermined changes in composition, and possibly because of folds and crypts in the mucous membrane, particularly near the vesicle neck, where the bacteria may be relatively protected. Whatever the explanation, clinical experience indicates the wisdom of stopping bacteriophage treatment when the patient has become free from symptoms and the bacterial count in the urine remains persistently below 20,000 per cubic centimeter for several days without complete sterility being obtained. One may sometimes attain final success in such instances by introducing silver nitrate in a 1 : 10,000 solution in distilled water into the urinary bladder on two successive days and following this by oral administration of methenamine and acid phosphate for two or three weeks.

Septicemia due to the colon bacillus is not common. We have had opportunity to treat only four patients with this condition in the past three years. To all of these the asparagine preparation of the bacteriophage was given intravenously, beginning with a small dose. To the three patients who survived, no dose larger than 5 cc. was given at any one time. One of these patients showed a shockingly severe reaction; another, only a slight rise in temperature, and the third, a mild chill following the intravenous therapy. The patient who died gave no reaction to the bacteriophage at any time, although the dosage was increased to 10 cc. of the undiluted preparation every two hours.

Inflammations of the large intestine which can be recognized as due to such organisms as the dysentery bacillus of Shiga have been treated by d'Herelle and others with bacteriophages with conspicuous success. Our own experience has related chiefly to stubborn colitis of long standing and uncertain etiology and to fecal fistulas persisting after surgical operations. We have not been able to recognize, in a satisfying way, the specific identity of the microbes concerned in causing the irritation

in these patients. The use of bacteriophages has, therefore, been empirical. However, one may justify a trial of bacteriophages here because their introduction into the digestive tract with the food and by colonic tube appears to be without serious untoward results and is sometimes followed by improvement which can hardly be accounted for by psychic impression alone. Ordinarily a bacteriologic study of the feces should first be made. The bacteriophages to be used are selected after test against the bacteria found. The broth-bacteriophage preparations are used, and usually several are mixed together so that the mixture will act against colon, dysentery and pyocyaneus bacilli and also staphylococci. Possibly the favorable results may be due to a partial suppression of various bacteria which are acting as secondary invaders of the lesions in the intestinal mucous membrane, thus permitting the natural defenses of the body to cope more successfully with the essential cause of the disorder.

In the succeeding papers of this series we purpose to present in more detail the records of a few patients suffering from infections with colon bacilli and to indicate the mode of application of bacteriophage therapy in each case.

SUMMARY

The preparation of bacteriophages active against colon bacilli presents particular problems, apparently because of the complex nature of the reactions between the bacteria in this group and the multiple bacteriophage agents to be considered.

Specific bacteriologic diagnosis and isolation in culture of the offending microbes should generally precede the therapeutic use of bacteriophages. A specific bacteriophage filtrate prepared with the infecting organism should be substituted at the earliest possible moment for any stock preparation which may have been employed as an emergency measure.

The essential conditions for most effective bacteriophage action at the site of the infectious lesion should be intelligently created and maintained.

Particular technical methods of clinical application for individual patients will be indicated in succeeding papers.

PERFORATION OF JEJUNAL ULCER INTO THE FREE ABDOMINAL CAVITY

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Jejunal (including gastrojejunal) ulcers of peptic origin are conceded to be relatively serious as compared with similar lesions in the stomach and duodenum. The seriousness of a jejunal ulcer can be attributed chiefly to the proclivity of the defect to extend rapidly beyond the wall of the intestine. This tendency is due to the thinness of the jejunum and to the inherent susceptibility to peptic corrosion of this portion of the digestive tract. The complications which develop as a result of the extension of an ulcer beyond the serous coat of the jejunum are of two principal types, penetrative and perforative. Penetration occurs when an adjacent structure becomes attached to the base of the ulcer before actual rupture occurs. Often there develops a granulomatous mass of considerable size simulating a neoplasm. Perforation generally occurs into the free peritoneal cavity or into a neighboring hollow viscus. Occasionally, however, the entire thickness of the ventral abdominal wall is perforated, and a communication between the jejunum and the exterior is established. When the rupture occurs into a hollow viscus it is the colon that is almost invariably involved. The subject of jejunocolic fistula resulting from a perforated ulcer has been extensively studied and accorded great attention in recent medical literature (Brams and Meyer,[1] Verbrugge,[2] Flörcken and Steden,[3] Urrutia,[4]

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1. Brams, W. A., and Meyer, K. A.: Diagnosis and Treatment of Gastro-Jejunocolic Fistula, Surg., Gynec. & Obst. **38**:646, 1924.

2. Verbrugge, J.: Gastrojejunocolic Fistulas, Arch. Surg. **11**:790 (Nov.) 1925.

3. Flörcken, H., and Steden, E.: Beiträge zur Entstehung und Therapie des Ulcus pepticum jejuni (U. p. j.) nach Magenoperationen nach einigen Erfahrungen und einer Umfrage bei 22 Chirurgen, Arch. f. klin. Chir. **143**:173, 1926.

4. Urrutia, L.: Das Ulcus pepticum jejuni nach Magenoperationen in Spanien, Arch. f. Verdauungskr. **40**:339, 1927.

Lahey and Jordan [5] and Sokolov [6]). On the other hand, rupture of a jejunal ulcer into the free abdominal cavity has not been the subject of a comprehensive report for many years. We deemed it desirable therefore, to investigate anew the free perforation of a jejunal ulcer, and to attempt to bring the literature up to date. As a result of this study we are able to confirm many of the statements made by previous writers and to add certain observations which we believe merit presentation. No attempt will be made to cover phases of the subject adequately treated in previous publications.

INCIDENCE

It is universally conceded that the free perforation of a jejunal ulcer is not of common occurrence. However, whether the condition is rare or merely uncommon appears to be an open question. The German literature particularly leads one to assume that the free perforation of a jejunal ulcer is almost a unique happening. Spath,[7] whose report appeared in 1927, stated that he could find accounts of only eleven perforated jejunal ulcers, three of which occurred in one patient. Spath's case of ruptured jejunal ulcer brought the number to twelve. In the statistical study by Fromme,[8] published two years later (1929), three cases were added, one by Porzelt [9] and two by himself. In the same year Just [10] recorded the results of his own review of the literature gathering seventeen cases of free perforation of a jejunal ulcer and adding one of his own. The latest report of a ruptured jejunal ulcer published in German periodicals is that of Steden [11] who, in 1931, described four cases in only three of which there was actual perforation. This brings the total, according to German writers, to twenty. This astonishingly small number is explained by the failure of the German writers to include cases reported in other languages. The figures of British authors, which embrace the cases of all countries, are much

5. Lahey, F. H., and Jordan, S. M.: Gastrojejunal Ulcers and Gastrojejunocolic Fistulae, Ann. Surg. **87**:231, 1928.

6. Sokolov, S. E.: Beiträge zur Frage des Ulcus pepticum jejuni postoperativum auf Grund eines Materials von 134 Fällen russischer Chirurgen, Arch. f. klin. Chir. **149**:230, 1928.

7. Spath, F.: Ueber das in die freie Bauchhöhle perforierte Ulcus pepticum jejuni, Deutsche Ztschr. f. Chir. **205**:113, 1927.

8. Fromme, A.: Ueber primäre Resektion des in die freie Bauchhöhle geplatzten Ulcus pepticum jejuni, Beitr. z. klin. Chir. **147**:111, 1929.

9. Porzelt, W.: Das perforierte Ulcus pepticum jejuni im Gefolge des Zwölffingerdarmgeschwürsdurchbruchs, Zentralbl. f. Chir. **55**:1740, 1928.

10. Just, E.: Zur Frage des frei in die Bauchhöhle perforierten Ulcus pepticum jejuni, Wien. klin. Wchnschr. **42**:500, 1929.

11. Steden, E.: Ueber Ulcus pepticum jejuni mit Perforation in die freie Bauchhöhle, Deutsche Ztschr. f. Chir. **230**:404, 1931.

more representative of the true incidence of jejunal perforation. In 1909 a comprehensive article by Paterson,[12] of London, on jejunal ulcers appeared in the *Annals of Surgery,* which included summaries of nineteen cases of perforation into the free abdominal cavity. In 1918 Wright,[13] of Manchester, collected thirty-two cases and Massie,[14] of London, in 1924, increased the number to thirty-seven. In each of two patients jejunal perforation recurred, bringing the total number of perforations to thirty-nine. We have been able to add to Massie's number fifty-eight perforations occurring in forty-nine patients (text at the end of the article). The bulk of these additional reports appeared after Massie's article was written, although a few were published between 1920 and 1924. This total of ninety-seven established instances of the free perforation of a jejunal ulcer affecting eighty-six persons to which four from our own records are added, indicates that the involvement, although relatively uncommon, cannot be considered actually rare. Subsequently we shall endeavor to show that in addition to the established cases there is a relatively large group of unrecognized free jejunal perforations which, when taken into account, materially raises the incidence.

INCREASED DISPOSITION OF RUPTURE AFTER JEJUNAL PERFORATION

Persons in whom rupture of a jejunal ulcer into the free abdominal cavity occurs appear to be particularly predisposed to perforation in general. One indication of this tendency is the striking frequency with which previous gastroduodenal rupture occurred in the case of jejunal perforations which we found reported. Of the forty-nine patients added to those listed by Massie [14] the nature of the original lesion was obtained in twenty-three. In fifteen (66 per cent) of these twenty-three the initial operation was performed for a ruptured gastroduodenal ulcer. In one of the remaining eight a perforated ulcer of the stomach was the cause of the patient's fourth gastric operation, the fifth and final one being a resection for jejunal rupture. It is more than likely that statistics based on a larger number of jejunal perforations would fail to give as high an incidence of previous gastroduodenal rupture. Nevertheless, the frequency of antecedent perforation in the stomach and duodenum in the small group studied cannot be disregarded. It signifies that in at least a fair percentage of gastroduodenal ruptures treated by gastro-enterostomy subsequent jejunal perforation is to be anticipated. On account of the frequent development of jejunal perforation

12. Paterson, H. J.: Jejunal and Gastrojejunal Ulcer Following Gastrojejunostomy, Ann. Surg. **50**:367, 1909.

13. Wright, G.: Secondary Jejunal and Gastro-Jejunal Ulceration, Brit. J. Surg. **6**:390, 1919.

14. Massie, G.: Perforated Jejunal Ulcer, Guy's Hosp. Rep. **74**:70, 1924.

several authors (Spath,[7] Frömme[8] and Brandtner and Tönnis[15]) recommend, when possible, the avoidance of simple gastro-enterostomy in the treatment of acute gastroduodenal perforation.

Another indication of a heightened predisposition to perforation in patients with jejunal rupture is the tendency toward recurrent perforation. Repeated jejunal ruptures occurred in six of thirty of the cases collected by us. In the remaining nineteen insufficient data were available for analysis. In Robinson's[16] patient a jejunal perforation occurred twice at intervals of about six months. Brandtner and Tönnis[15] performed a gastro-enterostomy for a perforated duodenal ulcer in a patient who subsequently had two jejunal perforations four months apart. Nixon and Lowry[17] operated on a patient on July 3, 1921, for an ulcer on the greater curvature, seen in a roentgenogram. A posterior gastro-enterostomy was performed. In October, 1922, the first perforation, located in the stomach just above the stoma, occurred. In March 1926, a jejunal perforation took place and in August 1927, a rupture similar to the first. A fifth operation, in December 1927, disclosed multiple gastrojejunal ulcers "two about to perforate in the colon." The maximum number of true jejunal perforations occurred in the case of Riess.[18] In conjunction with resection of a penetrating ulcer he performed an anterior gastro-entero-anastomosis. Three consecutive perforations of the jejunum took place on Sept. 27, 1922, March 27, 1923 and March 28, 1925, respectively. Irrespective of the location, the largest number of perforations of a peptic ulcer occurred in the cases reported separately by Davenport[19] and Henry.[20] Davenport's patient was first operated on for a perforated duodenal ulcer, at which time a posterior gastrojejunostomy was performed. Subsequently a jejunal perforation occurred twice, and the anastomosis was undone. Two additional gastroduodenal ruptures followed, bringing the total to five. Henry's patient had the same number of perforations. The first was gastric, the two subsequent were jejunal, the fourth was not specified and the fifth was located at the stoma.

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15. Brandtner, C. E., and Tönnis, W.: Beitrag zur Häufigkeit und Behandlung des Ulcus pepticum jejuni nach Gastroenterostomie wegen perforiertem Magen- und Duodenalulcus, Deutsche Ztschr. f. Chir. **236**:93, 1932.

16. Robinson, V. P.: A Case of Partial Gastrectomy for Pyloric Ulcer, Followed by Two Jejunal Ulcers Perforating at Intervals of Six Months: Recovery, Lancet **1**:703, 1928.

17. Nixon, P. I., and Lowry, S. T.: Multiple Consecutive Perforated Gastrojejunal Ulcers, with Report of a Case, M. J. & Rec. **128**:584, 1928.

18. Riess, P.: Zur Perforationsneigung des Ulcus Pepticum, Zentralbl. f. Chir. **52**:2818, 1925.

19. Davenport, G. L.: Five Operations in Twelve Years for Perforation, J. A. M. A. **97**:99 (July 11) 1931.

20. Henry, C. K. P.: Recurrent Gastric Perforations, Surg., Gynec. & Obst. **32**:542, 1921.

An additional manifestation of an increased susceptibility to perforation in the group of forty-nine patients with fifty-eight ruptures is found in the occurrence of six free perforations of the jejunum following gastric resection. Half the resections were wide; the other half were limited to a pylorectomy. In the case of Brandtner and Tönnis [15] an anterior gastro-enterostomy was performed during an operation for a perforated duodenal ulcer. A jejunal perforation followed, on which account the stomach was widely resected. Following recovery from the resection there occurred a jejunal perforation which was treated successfully by simple suture. Brütt's [21] patient also had a gastro-enterostomy performed following the perforation of a gastroduodenal ulcer. A year later an extensive resection of the Billroth II type was performed on account of symptoms caused by an unruptured jejunal ulcer. A year after the resection an acute perforation of a large, calloused jejunal ulcer developed for which operation was undertaken with fatal outcome. Postmortem examination disclosed two additional jejunal ulcers. Flörcken and Steden [3] described a case in which a gastrojejunal ulcer developed following a pyloric resection. An extensive resection was therefore performed, following which a second jejunal ulcer developed and subsequently perforated. In January 1927, Robinson [16] performed a Billroth II operation on the order of a pylorectomy for the cure of duodenal ulcer. The patient was free from symptoms until August 4, when an ulcer of the jejunum perforated. The operation consisted of suturing the ulcer and covering it with omentum. On Jan. 28, 1928, a second perforation of the jejunum distal to the anastomosis occurred. Operation was again followed by recovery. The sixth jejunal perforation in this group occurred in Steden's [11] patient who was operated on for the first time in 1922 when a perforated pyloric ulcer was closed. The patient was subsequently operated on several times, and finally in December 1922, resection and posterior gastro-enterostomy were performed. Four years later a gastro-jejunocolic fistula developed and subsequently a jejunal ulcer perforated into the free abdominal cavity.

ACUTE PERFORATION OF JEJUNAL ULCER WITH SPONTANEOUS CLOSURE

Benign ruptures occurring in connection with the stomach and duodenum have been recognized for many years. These were generally referred to as "subacute" perforations (Lund [22] and Moynihan [23]). It

21. Brütt, H.: Bericht über die 34 Tagung der Vereinigung Nordwestdeutscher Chirurgen, Zentralbl. f. Chir. **54**:2268, 1927.

22. Lund, F. B.: Subacute Perforation of the Stomach with Report of Three Cases, Boston M. & S. J. **152**:516, 1905.

23. Moynihan, B. G. A.: Subacute Perforation of the Stomach and Duodenum, Ann. Surg. **45**:223, 1907.

was generally assumed that on account of the leisurely manner in which the destruction of the gastroduodenal wall occurred neighboring adhesions had an opportunity to form prior to actual rupture. The presence of these adhesions presumably led to the formation of a sac which, when perforation took place, prevented the escape of gastroduodenal content into the general peritoneal cavity. It was thought that the leakage and exudate were restricted to the preformed sac and either were absorbed or underwent suppuration leading to the development of a perigastric abscess. In 1912 Schnitzler [24] offered convincing evidence that at least in certain cases of perforation with a benign course rupture into the free peritoneal cavity actually occurred, but that shortly following perforation the opening was covered by a neighboring structure, usually the liver. According to Schnitzler, therefore, the adhesions which form in connection with so-called subacute ruptures result from and follow rather than precede perforation.

Singer and Vaughan [25] confirmed Schnitzler's conception, employing among other methods roentgenography. They found that in cases of so-called subacute perforation of the stomach and duodenum the escape of air occurs not into a perigastric pocket but rather into the free peritoneal cavity. The bubble of gas seen fluoroscopically can be made to shift to all portions of the abdomen with a change in the position of the patient. It was pointed out that in these cases of benign perforation a minute-by-minute history reveals the occurrence of sudden, violent pain identical with that of the classic rupture. Evidence was offered to show that the initial signs of subacute perforation are characteristic of diffuse peritonitis, and that the chief differences between the classic and the benign ruptures are observed subsequent to the initial manifestations, i. e., in the postperforative stage. Whereas in the classic rupture the annihilating pain of onset is followed by symptoms and signs of progressive peritonitis which does not remit until death, in the so-called subacute type the diffuse peritonitis of onset rapidly recedes, and spontaneous recovery ensues. If a patient with a benign rupture is seen for the first time several hours after the onset the manifestations are generally those of mild, retrogressing, local peritonitis. In order to emphasize the incomplete character of the clinical picture in the benign cases as compared with that of a classic perforation Singer and Vaughan suggested the term "forme fruste." The mild postperforative course in these cases is explained on the basis of early spontaneous closure of the gastroduodenal aperture.

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24. Schnitzler, J.: Ueber gedeckte Magenperforationen und über die Entstehung der penetrierenden Magengeschwüre, Med. Klin. **1**:938, 1912.

25. Singer, H. A., and Vaughan, R. T.: The "Formes Frustes" Type of Perforated Peptic Ulcer, Surg., Gynec. & Obst. **50**:10, 1930.

Theoretically it is to be expected that the forme fruste type of rupture should occur in connection with jejunal ulcer, just as it does with gastroduodenal ulcer. That free perforation of a jejunal ulcer followed by early spontaneous closure does take place is borne out by our own experience (cases 2, 3 and 4). Regarding the incidence of forme fruste jejunal ulcers, however, little of value can be said based on the literature. It is possible that many jejunal ulcers considered to be penetrating had their origin in unrecognized perforations which sealed spontaneously. Particularly does this appear plausible when one considers the history of forme fruste perforations affecting the stomach and duodenum. Until recently it was generally assumed that benign gastroduodenal perforations were exceptional. After learning how to recognize forme fruste ruptures and instituting a search for them, one of us (H. A. S.) [26] observed forty instances in a period of eighteen months in one, albeit large, hospital. Subsequent experience has demonstrated that the period of observation was not unusual from the standpoint of the number of cases encountered. If the manifestations of a forme fruste jejunal perforation become generally recognized and if the possibility of its occurrence is kept in mind, the present concept of the incidence of free perforation of this type of ulcer may be altered considerably.

REPORT OF CASES

CASE 1.—*Perforation of jejunal ulcer into free abdominal cavity; operative closure; recovery.*

K. M., a white woman, 42 years of age, was admitted to the University Hospital on July 10, 1930, with the history that for eight years prior to 1929 she had experienced attacks of burning epigastric pain which occurred about two hours after meals and which was completely relieved by soda, milk or vomiting. In 1929 the distress was associated with persistent vomiting which did not submit to medical management. Pyloric stenosis had apparently developed, and a gastroenterostomy was done elsewhere. The patient obtained complete relief from her symptoms, and was instructed to remain on a soft diet. About three months previous to admission she tired of the diet and ate freely of irritating foods. In spite of dietary indiscretions she had no distress until about a week before entrance when she noted bloating and nausea about a half hour after meals, followed at times by vomiting of material resembling coffee-grounds. She experienced no pain similar to that originally suffered, or any new pain left of the midline.

On the day of admission the patient was free from symptoms until 10 a. m. when she was suddenly seized by severe epigastric pain which caused her to double up. The pain rapidly spread from the epigastrium to the lower part of the abdomen, and within a few minutes of the onset it became intolerable. Vomiting occurred shortly after the initial occurrence of pain, the vomitus having the appearance of coffee-grounds. The pain continued unabated until the anesthetic was administered. Physical examination at the time of admission disclosed essentially diffuse abdominal tenderness and boardlike rigidity. The dulness of the liver was not obliter-

26. Singer, H. A.: Spontaneous Recovery from Perforation of Peptic Ulcer into the Free Abdominal Cavity, Arch. Int. Med. **45**:926 (June) 1930.

ated, and no peristalsis was heard on auscultation. Siphonage of the stomach yielded 1,000 cc. of a material resembling coffee-grounds. The white blood cell count was 12,000. The temperature, pulse rate and respiratory rate were relatively normal. A diagnosis of ruptured peptic ulcer was made, and immediate operation advised.

On opening the abdomen, under ether anesthesia, a milky exudate escaped. Exploration of the stomach and duodenum revealed no evidence of a perforation. The stoma of the posterior gastro-enterostomy was then exposed, and a perforation about 2 mm. in diameter was found on the anterior wall of the jejunum 1 cm. distal to the site of anastomosis. The perforation was encircled by a moderate amount of induration. The opening was closed with three interrupted linen sutures, and an appendix epiploica, which was amputated from the transverse colon, was sutured over the site of perforation. The abdomen was closed without drainage. The patient made an uneventful recovery, and when last seen a year after the perforation was free from symptoms.

CASE 2.—*Perforation of a jejunal ulcer into the free abdominal cavity followed by formation of an abscess; operative drainage; recovery.*

B. G., a colored man, 38 years of age, first entered the Cook County Hospital on Aug. 4, 1928, on account of abdominal pain and vomiting. He had had symptoms typical of ulcer periodically since the age of 15. He was treated medically until 1915, when gastro-enterostomy and entero-enterostomy were performed. He remained well for twelve years, after which the symptoms recurred. In June, 1927, a second operation was undertaken, the gastrojejunostomy being undone and the stomach resected. The anastomosis was of the posterior Polya end-to-side type. The old entero-enterostomy was not disturbed. Following recovery the patient remained well for from six to seven months; then he began to be troubled by pain located below and to the left of the umbilicus. The pain, which was mild and localized at onset, soon increased in severity, and radiated through to the back. Food and the usual dose of sodium bicarbonate were ineffective in affording relief. At times during the attack of pain vomiting would occur, but would not be followed by cessation of pain. Morphine was frequently required for relief. Shortly after entering the hospital the patient had a profuse gastro-intestinal hemorrhage, passing several tarry stools and vomiting clotted blood. After institution of the Sippy treatment the pain disappeared, and the bleeding ceased. Roentgen examination undertaken several weeks after the hemorrhage failed to demonstrate an ulcerous niche in the remnant of stomach or in the stoma. The patient was discharged on Sept. 26, 1928, free from abdominal symptoms but still anemic.

He returned on Nov. 9, 1928, chiefly on account of symptoms referable to the persistent anemia. The pain below and to the left of the navel and in the back had returned, but was now relieved by the administration of alkalis. On November 11, two days after readmission, the patient was seized suddenly by intense periumbilical pain which rapidly spread to involve the lower part of the abdomen. He described the pain as knifelike and of far greater intensity than any pain he had previously experienced. Instead of continuously moving as he was accustomed to do in his previous attacks he remained absolutely immobile. Any attempt at change of position was associated with intense, stabbing pain. Deep breathing was also inhibited on account of resulting pain. The intern assumed that this attack of pain was similar in origin to the ones witnessed by him during the patient's first stay in the hospital. He therefore attempted gastric aspiration which seemed to aggravate rather than to relieve the pain. He then ordered morphine (¼ grain [16.2 mg.]) which afforded the patient partial relief. When

seen by an attending physician six hours after the onset of violent pain the patient was fairly comfortable. The attending physician noted a moderate amount of tenderness throughout the abdomen, with increased resistance, but no boardlike rigidity. There was little or no distention. The peristaltic sounds were decreased in number and quality. He suspected the possibility of a perforation, but hesitated to make the diagnosis because of the rapid improvement which followed the violent pain of onset and on account of the lack of classic signs. Each hour that passed found the patient greatly improved and the signs of peritonitis less definite.

We were first consulted five days after the onset of the initial violent pain because of the development of a temperature which ranged between 100 and 101 F.

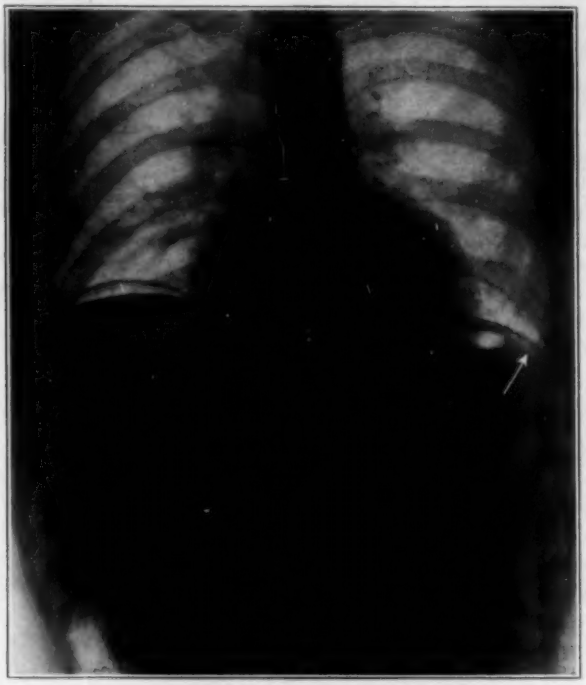

Fig. 1 (case 2).—Pneumoperitoneum due to the perforation of a jejunal ulcer. The sickle-shaped area of radiolucence beneath the diaphragm on the right side represents free air. On the left side a fluid level is seen due to liquid and gas in the stomach. Adjacent to the gastric air bubble is a small amount of free air (indicated by arrow).

On examination an ill-defined mass could be palpated in the lower part of the abdomen to the left of the midline. On the basis of the history, which was typical of acute rupture, we assumed the presence of a closed perforation with the development of an intra-abdominal abscess. Fluoroscopic examination established the diagnosis for practical purposes by demonstrating the presence of a pneumoperitoneum (fig. 1). Operation at this time was deemed inadvisable and was deferred until November 27, twelve days after the onset of the acute pain. The mass which had increased and become more definite was incised, and its purulent con-

tents drained. When the patient was discharged on Jan. 9, 1919, a small amount of drainage was still present, but the patient's general condition was good. He failed to return for further observation.

CASE 3.—*Perforation of a jejunal ulcer into free abdominal cavity in a patient with old gastrocolic fistula; spontaneous closure of recent perforation; recurrent leakage from rupture following heavy meal; operation; death.*

E. B. N., a white man, 50 years of age, was admitted to the Cook County Hospital on the evening of Dec. 23, 1929; the diagnosis made in the admission room was intestinal obstruction. The preliminary diagnosis apparently prejudiced the subsequent examiners, for the history as recorded by them indicates that they were governed by a preconceived idea. It was stated that the patient entered on account of severe abdominal pain of four hours' duration. The acute illness was preceded by symptoms of "colitis," present for a period of five months, and characterized by from twelve to fifteen evacuations a day. On the day of admission the patient ate a hearty breakfast at 8 a. m. At 10:30 a. m. he was seized with shooting pain which traveled across the middle of the abdomen, causing him to double up. The pain continued to be severe, and within thirty minutes had spread to involve the lower left quadrant, where it reached maximum intensity. The pain did not abate. No stool or flatus had been expelled since the onset.

The patient had been treated medically for peptic ulcer in 1919, and was operated on for ruptured ulcer in 1921. In 1923 a gastro-enterostomy was performed, and in 1926 the patient suffered a severe gastro-intestinal hemorrhage. Nothing was recorded regarding the character of symptoms which preceded the "colitis," nor was an attempt made to relate the diarrhea to the development of a gastrocolic fistula.

Physically the patient was described as suffering from severe intermittent pain. The presence of a cold sweat was noted. Abdominal examination disclosed two scars in the upper half, diffuse tenderness most marked on the left side and a firm tenseness of the abdominal wall which prevented palpation of the intra-abdominal contents. It was stated that peristaltic waves were not visible, but that increased peristalsis was heard on auscultation. A diagnosis of incomplete intestinal obstruction was made, although the possibility of a complication of the colitis was also considered. Enemas, gastric lavage and hypodermoclysis were instituted, following which the patient was more comfortable.

The next morning, December 24, the patient was given a barium sulphate enema which met obstruction at the sigmoid flexure, caused by what appeared to be an extrinsic mass. However, the pain had completely disappeared, and the patient was quite comfortable. On account of the decided clinical improvement operation was not deemed advisable at that time. On the following day, December 25, the patient was served Christmas dinner at noon. In the afternoon he complained of severe abdominal pain similar to that experienced on the day of onset. The pain moderated after several hours, but nevertheless was felt keenly after that time. During the next two days distention appeared, and accompanied the pain.

It was not until December 28, six days after admission, that we were consulted regarding the patient. A careful perusal of the history was sufficient to suggest the probable diagnosis. The "colitis" which was present for five months and which followed the gastro-enterostomy was considered to be the result of a gastrojejunocolic fistula. The sudden, intense pain which began four hours prior to admission was assumed to be due to a perforated jejunal ulcer. The subsidence of peritonitic manifestations was ascribed to spontaneous closure of the perforation. The recurrence of acute symptoms was attributed to reopening consequent to the

ingestion of the Christmas dinner. Since the patient was not improving and the possibility of a patent perforation could not be excluded, operation was recommended and undertaken immediately.

At operation the peritoneal surfaces were found to be covered with a fibrinous exudate. After wiping away the fibrin from the region of the stoma, at which point it was thickest, a perforation the size of a pinhead was exposed 4 cm. distal to the anastomosis on the anterior wall of the jejunum. The perforation was readily sutured. The colon was intimately attached to the stomach with which it communicated in the region of the greater curvature. The posterior gastro-enterostomy was undone in order to repair effectively the gastrocolic fistula. Since an

Fig. 2 (case 4).—Roentgenogram taken five hours after the administration of a barium meal. The retention in the stomach and duodenum was due to the obstruction of the proximal jejunum by the compression of an abscess following perforation of a jejunal ulcer.

obstruction existed at the duodenum it was necessary to perform another gastroenterostomy. The day following operation signs of spreading peritonitis developed, and the patient died on December 30, two days following operation and one week after the onset of the perforation.

CASE 4.—*Perforation of jejunal ulcer into free abdominal cavity; development of high intestinal obstruction due to compression by complicating abscess; incision and drainage of abscess; recession of obstruction and recovery.*

L. H., a white man, 49 years of age, was referred to one of us (K. A. M.) on Sept. 4, 1931, for surgical relief from a duodenal obstruction. The history sub-

mitted by the referring physician stated that the patient began to suffer in 1921. The symptoms of an ulcer continued intermittently until 1924, when a posterior gastro-enterostomy and resection of the ulcer were performed. Relief was obtained for a continuous period of four years, after which an attack of epigastric pain and vomiting occurred which lasted only a few days. The patient remained well until two weeks before, when he experienced another recurrence. In spite of strict medical management the symptoms failed to submit to control, and on this account roentgen examination was proposed. A barium sulphate meal administered two days before our first examination disclosed a dilated stomach with hyperperistalsis

Fig. 3 (case 4).—The stomach and upper intestinal tract shown in figure 2 after the patient had recovered from the incision and drainage of the parajejunal abscess. The exposure was made three hours after the radiopaque meal was ingested, and demonstrates an absence of the previous duodenal obstruction.

and a greatly dilated duodenum which was apparently obstructed (fig. 2). The cause of the obstruction was assumed to be a gastrojejunal ulcer which produced narrowing of the stoma.

Our associate, Dr. Rosi, in eliciting a careful history obtained a classic account of an acute perforation which took place at the time of onset of the alleged recurrence. The intolerable initial pain, which was diffuse, gradually abated, and was replaced by a dull ache located in the left upper quadrant of the abdomen. Nausea and vomiting developed later, and became more and more persistent. Physical examination two weeks following the acute perforation led to the discovery of a

tender mass, the size of a lemon, just above the level of the umbilicus to the left of the midline. The temperature was 101.6 F., and the white cell count was 22,400. The diagnosis of perforated jejunal ulcer with subsequent formation of an abscess was made, and the obstruction attributed to the associated collateral inflammatory edema. It was thought advisable to treat the patient conservatively in the hope that the inflammation would subside and the obstruction recede. After withholding ingesta for a week and furnishing fluids, salt and dextrose by venoclysis the mass and tenderness disappeared, and the temperature and white cell count returned to approximately normal. The patient was then given a liquid diet, and appeared to be doing well for six days, but on the following day he vomited 1,500 cc. of material. Operation was therefore decided on, and performed on September 19.

After reflecting the transverse colon upward and exposing the site of the gastroenterostomy an abscess was encountered lying between the angle formed by the afferent and the efferent loops. The abscess compressed and narrowed the adjacent bowel, and was apparently responsible for the obstruction. The gastro-enterostomy stoma was widely patent, having a breadth of three fingers. The pus was evacuated and the cavity of the abscess drained. No attempt was made to search for the jejunal perforation. The convalescence was uneventful, and the patient was discharged feeling well. Roentgen examination undertaken following recovery from operation showed no dilatation of the stomach or duodenum, but a poorly functioning gastrojejunal stoma (fig. 3).

COMMENT

The account of case 1 is a classic description of a jejunal perforation into the free abdominal cavity. It is typical of the clinical picture which characterizes the cases reported in the literature. In the other three cases jejunal perforation occurred likewise into the free abdominal cavity. However, the clinical picture was different, and careful analysis was necessary to establish the fact that acute rupture had occurred. In the second case an intra-abdominal abscess was traced to an acute perforation which was not recognized as such by the attending physician who saw the patient early. The lack of recognition of the rupture was due to the spontaneous closure of the perforation and the limitation of jejunal leakage. The evidence in this case for perforation into the general abdominal cavity rather than into a preformed sac was afforded by fluoroscopy which demonstrated that the escaped air was free. The genesis of the abdominal abscess is presumably the same as in perigastric abscess following rupture of the stomach or duodenum. It has been shown by Singer and Rosi [27] that intraperitoneal accumulations of pus complicating gastroduodenal perforation are the result of encapsulation of exudate following and not preceding rupture.

In the third case the patient was thought to be suffering from an intestinal obstruction. However, the history indicated clearly that the

27. Singer, H. A., and Rosi, P. A.: The Pathogenesis of Perigastric Abscess Complicating Peptic Ulcer, Am. J. M. Sc. **183**:600, 1932.

symptoms and signs at the outset were those of diffuse peritonitis, and that localization did not occur until some time later. Apparently spontaneous closure of the opening in the jejunum occurred shortly following rupture, and the escaped material was absorbed by the peritoneum. A second attack of severe pain two days subsequent to the first occurred following the intake of a heavy meal. Here also the manifestations were those of diffuse peritonitis due presumably to the separation of fibrinous adhesions and the reopening of the perforation. The bewildered clinicians were unable to reconcile the clinical history with the physical observations. As the operation subsequently demonstrated, sealing of the perforation was accomplished by nature following the second rupture also. However, the closure of the aperture was not effected soon enough or was not sufficiently secure to prevent the escape of enough material to produce moderately severe peritonitis. The confusing element in the case was the intermittent leakage, a phenomenon which has been observed previously in connection with gastroduodenal perforations (Singer [28]). Death might have been averted if the surgical procedures had been limited to the acute aspects of the case, and the old gastrocolic fistula had been dealt with at a more favorable time. When first seen, the patient in the fourth case presented roentgenologic and clinical evidence of a high intestinal obstruction. The history led to the diagnosis of a recent free perforation of a jejunal ulcer complicated by the formation of an abscess. The pathogenesis of the abscess was presumably similar to that complicating the rupture in case 2.

It is difficult to estimate the incidence of free perforation of jejunal ulcer followed by spontaneous closure. In the literature dealing with rupture of jejunal ulcer an occasional reference (Urrutia,[4] Brütt [29]) is made to the occurrence of a "covered" perforation. It appears, however, particularly in the case of Brütt, that the adhesions which were found about the base of the ulcer might have preceded rather than followed the destruction of the serosa. The lack of published reports on the free perforation of the jejunum with spontaneous closure would seem to indicate that this sequence is rare. Our own experience, unless unique, would signify otherwise. It is more than likely that some cases of jejunal ulcer characterized by severe pain and diagnosed as penetration or subacute perforation represent acute ruptures with subsequent spontaneous sealing of the hole. Careful attention should

28. Singer, H. A.: Perforated Peptic Ulcer with Intermittent Leakage, J. A. M. A. **102**:112 (Jan. 13) 1934.

29. Brütt, H.: Beiträge zur Klinik und zur operativen Behandlung des peptischen Jejunalgeschwürs nach Gastroenterostomie, Beitr. z. klin. Chir. **126**:41, 1922.

be paid to the details of onset and the subsequent developments in patients on whom gastro-enterostomy has been done and who experience severe abdominal pain. A proper analysis of such cases, plus a knowledge of the mechanism of a forme fruste rupture may lead to the realization that the free perforation of jejunal ulcer occurs relatively oftener and that many free perforations pass unrecognized.

SUMMARY

A statistical survey of the literature on the perforation of jejunal ulcer into the free abdominal cavity yields a total of ninety-seven instances occurring in eighty-six persons. A striking feature in patients who have a rupture of a jejunal ulcer is the increased disposition toward perforation. The enhanced perforative tendency is indicated by the frequency of previous gastroduodenal rupture and recurrent jejunal perforation even following resection.

Four cases of rupture of jejunal ulcer into the free peritoneal cavity are reported. The first example is of the classic variety, and is typical of the cases reported in the literature. The other three instances are atypical in that two were characterized by the formation of an abscess and the third by evidence of intermittent attacks of perforative peritonitis. In analyzing carefully the two cases with abscesses, the initial symptoms were found to be characteristic of diffuse peritonitis. The clinical manifestations indicated primary free perforation with spontaneous closure of the hole and localization of the exudate. In one case the presence of a movable intraperitoneal bubble of gas served to substantiate the opinion that the rupture occurred into the free abdominal cavity. In the case with intermittent symptoms the initial perforation was of the forme fruste type; that is, the rupture was accompanied by a minimum amount of leakage, owing to prompt, spontaneous sealing of the aperture. A hearty Christmas dinner was followed by a recurrence of symptoms presumably due to the separation of the fibrinous adhesions and reopening of the hole. With better appreciation of the manifestations of free, acute perforation of a jejunal ulcer followed by spontaneous closure the diagnosis of penetration or "subacute" perforation will not be made as often as heretofore. Furthermore, it is believed that the recognition of atypical cases will indicate that the free perforation of a jejunal ulcer is not uncommon.

RECENT CASES REPORTED IN THE LITERATURE

The following list does not include the thirty-four cases collected by Massie.[14] The number of cases recorded by each author is indicated in the figure or figures preceding each name.

1-2. Ashcroft, A. T.: Two Cases of Perforated Jejunal Ulcer, Brit. M. J. **1**:528. 1926.

3. Brandtner and Tönnis.[15]

4. Brütt.[21]

5. Bundschuh: Zur Frage der Resektion beim freiperforierten Magen-Zwölffingerdarmgeschwür, Arch. f. klin. Chir. **129**:281, 1924.

6. Davenport.[19]

7. Ehrlich, S. P.: Repeated Perforations of Peptic Ulcers with Recovery, J. A. M. A. **90**:1870 (June 9) 1928.

8. Flörcken and Steden.[3]

9-11. Fromme.[8]

12. Henry.[20]

13. Jura, V.: Perforation of Jejunum Following Gastro-Enterostomy for Duodenal Ulcer, Boll. e atti d. r. Accad. med. di Roma **53**:131, 1927.

14. Just, E.: Zur Frage des frei in die Bauchhöhle perforierten Ulcus pepticum jejuni, Wien. klin. Wchnschr. **42**:500, 1929.

15-16. Kunz, H.: Ueber das perforierte Magen-Duodenalgeschwür und das perforierte pepticum jejuni, Arch. f. klin. Chir. **140**:419, 1926.

17-18. McKelvey, J. L.: Perforated Gastro-Jejunal Ulcer, M. J. Australia **2**:414, 1922.

19. Nixon and Lowry.[17]

20. Porzelt.[9]

21. Recht, H.: Aeussere Spontanfistel bei postoperativem Ulcus pepticum jejuni, Deutsche Ztschr. f. Chir. **224**:344, 1930.

22. Riess.[18]

23. Roberts, A. T.: A Case of Perforating Jejunal Ulcer Following Posterior Gastro-Enterostomy, M. Press & Circ. **109**:480, 1920.

24. Robinson.[16]

25-27. Schwarz, E.: Ueber die operative Behandlung des perforierten Magen- und Duodenalgeschwürs und der Perforation des peptischen Jejunalulcus nach der Gastroenterostomie, Deutsche Ztschr. f. Chir. **192**:239, 1925.

28-30. Sokolov.[6]

40. Spath.[7]

41-43. Steden.[11]

44. Steinberg, M. E.: Acute Perforation of Jejunal Ulcer, Northwest Med. **23**:187, 1924.

45-47. Urrutia.[4]

48. Winkelbauer, A.: Zur Frage des postoperativen Ulcus pepticum jejuni, Arch. f. klin. Chir. **140**:427, 1926.

49. Zeno, A.: Perforation of Jejunal Ulcer After Operation on Perforated Duodenal Ulcer, Rev. méd. del Rosario **1**:150, 1926.

NOTE.—Since the present article was submitted for publication a fifth case of perforated jejunal ulcer has been observed by us:

A man, aged 43, had suffered from symptoms of ulcer since 1903. Eventually evidence of pyloric obstruction appeared, on which account a gastro-enterostomy was performed in 1920. On Jan. 10, 1934, he suffered from acute perforation of an ulcer located at the stoma. At operation, performed eleven hours after the initial pain of rupture, the aperture was found to be concealed by fibrin. Suture of the perforation was followed by uneventful recovery.

PEPSIN IN THE PREVENTION OF ABDOMINAL ADHESIONS

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There are many controversies concerning the use or abuse of methods for the prevention of adhesions, and sides are taken by men of equal renown. Favoring the advancement of the study of prevention of adhesions are men who have devoted much time to experimentation to prove or disprove certain theories. Among them are Payr, Vogel and Siderenko, and more recently Kubota, Ochsner, Warren and Trusler. On the other hand, some have abandoned all hope of finding a substance which will take nature's place, and most prominent among them are Hertzler, D. F. Jones and W. L. McClure. The subject is concrete and lends itself to experimentation.

The purpose in view in all work of this type is not so much to prevent entirely the formation of all adhesions as to limit their formation to such an extent that they may not result in complications. It is not the adhesions, but the complications resulting from them which account for the fatalities attributed to them. Adhesions are desirable to a certain extent, for without them there would be innumerable postoperative fatalities. They constitute a natural process of protecting the peritoneal cavity from the spread of infection, and thereby limit the absorption of toxins when these are present. When no infection is evident, they represent nature's method in the healing process, but frequently they reach such proportions as to be not only undesirable, but dangerous.

In 1923 Deaver [1] classified adhesions as congenital and acquired; of the latter group he mentioned the inflammatory and operative types, but since injury to the peritoneum results in the same changes as an inflammatory process without infection we can classify these two as one. Of inflammatory adhesions he said that they may be constructive or destructive, which is analogous to Hertzler's [2] classification of physio-

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1. Deaver, J. B.: Intra-Abdominal Adhesions, Surg., Gynec. & Obst. **37**:506, 1923.

2. Hertzler, A. E.: The Peritoneum, St. Louis, C. V. Mosby Company, 1919, vol. 1.

logic and pathologic adhesions: physiologic if they degenerate after their function is performed, and pathologic if they become progressive and permanent. Physiologic adhesions are essential, when their need is urgent to help prevent the spread of infections. But, these adhesions take place early, sometimes within a few hours from the time trauma has been induced (MacCallum [3]), although the fibrin is deposited from ten to thirty minutes from the time the injury has taken place. The adhesions are expected to retrogress. Whether they do or not depends on several factors, among the most important of which are the individual tendency of the patient, too extensive trauma and the extent of the infection. There are certain adjuncts within the power of the operator which tend to limit the formation of adhesions, but these do not solve the problem. The solution lies, as Jones and McClure [4] put it, in the imitation of nature's method of prevention and ablation of adhesions. With this principle in view, the studies reported here deal especially with the biochemical action in the development and the retrogression of adhesions.

The manifestations of the process involved in the formation of adhesions will be modified by the anatomic surroundings and the physiologic activity of the peritoneum. It is necessary, therefore, to have a clear understanding of the anatomy, physiology and pathologic physiology of the peritoneum before one can undertake to alter the normal mechanism. The omentum is a peritoneal organ which plays a great part in physiologic activity in the process of repair. It is covered by a flat layer of endothelial cells, any injury to which calls forth great protective action on its part. The great rôle of the omentum is to wall off the site of an infection by adhering to it and thus preventing spread of the infection. It is well known that the omentum has a particular tendency to cling to roughened or infected surfaces. The parietal peritoneum lines the cavity and the visceral peritoneum covers the organs as a whole or in part, depending on their position and relation. Its function is primarily twofold: (1) to provide a smooth surface so that the organs may have free gliding movements during their normal activity, and (2) to absorb excess secretion in the cavity. Experiments have been performed to determine the route of the absorption with diphtheria toxin used as the agent. The conclusion was that the toxin was absorbed through the blood stream as well as through the lymphatic system. Though there is some controversy the route of absorption is irrelevant here. But what is of concern is that absorption takes place with remark-

3. MacCallum, W. G.: Text Book of Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1932.

4. Jones, D. F., and McClure, W. L., in Lewis, Dean: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1929, vol. 7, chap. 8, p. 49.

able rapidity. This is one of the factors to bear in mind when using solutions to inhibit the formation of adhesions. Experimental animals have absorbed as much as 8 per cent of their body weight in one hour. If the amount injected does not exceed 10 per cent of the body weight, 30 per cent or one third of the solution is absorbed in thirty minutes. The rate of absorption varies with the character and density of the fluid used, and may be delayed by peristalsis, because it is by peristalsis and the excursion of the diaphragm that the fluid injected is moved about. Since the rate of absorption is rapid, we therefore see that any substance introduced must be beneficial by virtue of its initial and rapid action as well as of its other properties.

During an insult to the peritoneum, by either chemical or mechanical means, there is an immediate deposit of fibrin on the surface. This process is comparable to an inflammatory reaction; it increases the tendency for adjacent surfaces to adhere and is responsible for the formation of fibrinous and then of fibrous adhesions which may eventually prove to be permanent and dangerous. The deposition of bundles of fibrin has been observed by Hertzler as early as ten minutes following the approximation of surfaces; the process is usually completed in two hours, and after six hours the adhesions are fairly firm. The rapidity with which these fibers are laid down is emphasized by the observation that during an operation for a gastro-enterostomy the first line of sutures is completely covered by fibrin by the time the operation is completed. The entire mechanism of the peritoneum is concerned with the attempt to localize and limit the extent of serosa involved.

Lacey [5] says that in the early reaction to an inflammatory process there is local leukocytosis, and that through the action of proteolytic ferments resulting from the leukocytosis the exudate which forms is absorbed. Consequently there is a dissolution of the products which results in adhesions. To duplicate this process artificially it would be necessary to devise some way of imitating nature's method and to bring about the resorption of those products the presence of which sometimes results disastrously for the patient and becomes a source of embarrassment to the surgeon.

The factors which increase the dangers of formation of adhesions are: (1) exposure and handling of viscera by the use of force, dry sponges and packing; (2) infection; (3) blood in the presence of an abrasion and a collection of blood in the cavity; (4) chemical irritation by iodine, iodoform and ether; (5) the use of a cautery; (6) sutures and ligatures; (7) subperitoneal hematoma, and (8) substances introduced for the prevention of adhesions.

5. Lacey, J. T.: Ann. Surg. **92**:281 (Aug.) 1930.

REVIEW OF THE LITERATURE

Some of the substances and methods used for the prevention of adhesions bring about the very thing which they were expected to prevent. Of the literature on this subject, the most logical and complete review is the outline of Ochsner and Garside,[6] in which they mentioned first the mechanical methods employed: the omental grafts suggested by Nicholas Senn in 1888 and peritoneal flaps. In 1894 the use of foreign substances was first introduced; among the first were fish bladder and the peritoneum of a calf, sterilized ox peritoneum or Cargile membrane, oiled silk and silver foil. Then a series of substances was used apparently for their lubricating effect: sterile olive oil, petrolatum and camphor liniment, the use of which was recommended by such authorities as Pfannenstiel, Hahne and Seuffert. Hydrous wool fat, U. S. P., a brand of thymol iodide, paraffin, acacia, gelatin mucin and albumin of egg were used. To demonstrate to what extreme science will go, reference is made to a Dr. Pribram who advocated the use of the vitreous humor of calves' eyes. Simultaneous with the use of these more or less solid substances, a series of experiments was carried on with the idea of keeping the serous surfaces apart, and in this group saline solution, citrate, dextrose and a combination of the last two were used. Among other methods mentioned are the use of oxygen in the cavity, high enemas, filling of the bladder, the administration of peptone and thiosinamine and stimulation of peristalsis. All of these methods were tried and in the final analysis were found wanting. More recently Johnson [7] reported his work on the use of amniotic fluid prepared in one of several ways. His results were excellent and were corroborated by Warren [8] and Trusler.[9] Lacey [5] repeated the experiments, but did not get the same results, although he admitted that the fluid might modify the density of the adhesions.

The final era in this type of work began with the use of digestive ferments in the prevention of abdominal adhesions. A number of investigations are recorded concerning the efficiency of these ferments.

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6. Ochsner, A., and Garside, Earl: Surg., Gynec. & Obst. **54**:338 (Feb.) 1932.

7. Johnson, H. L.: Amniotic Fluid Concentrate in the Prevention of Adhesions, New England J. Med. **199**:661, 1928; Observations on the Prevention of Post-Operative Peritonitis and Abdominal Adhesions, Surg., Gynec. & Obst. **65**: 612 (Nov.) 1927.

8. Warren, Shields: Effect of Amniotic Fluid on Serous Surfaces, Arch. Path. **6**:860 (Nov.) 1928.

9. Trusler, H. M.: Peritonitis: Experimental Study of Healing in the Peritoneum and the Therapeutic Effect of Amniotic Fluid Concentrate, Proc. Staff Meet., Mayo Clin. **4**:356 (Dec. 11) 1929.

Stewart [10] described an experiment which would make one believe that the action of pepsin results in the absorption or digestion of fibrin. Theoretically, then, it may be assumed that in this manner the fibrin which is formed on the irritated peritoneum is also digested by the pepsin. In 1922 Payr [11] first used a product which he called pepsin-Pregl solution which consisted of pepsin as the digestant and Pregl's solution, the latter being added to sterilize the ferment. Ochsner stated that Frankenthal corroborated the experiments and found that the substance inhibits the formation of adhesions. Kubota was unable to confirm this. Convinced of the futility of the numerous other substances, Kubota tried papain as the digestant of choice and obtained satisfactory results. With his results as an incentive Ochsner [12] used a solution of papain in various strengths and found that in solution ranging from 1 : 100,000 to 1 : 400,000 the activity was effective. He also used trypsin, but was not satisfied. He found that its action was inhibited by antitrypsin, which is normally found in exudates and transudates. He continued then with the papain, but encountered some difficulty in the solubility of the product, and the experimentation for a time was abandoned.

EXPERIMENTAL WORK

Continuing on the theory that the use of digestive ferments is rational, we decided to use an extract of pepsin, first in hydrochloric acid and then in glycerin, but free from iodine. We knew the powerful antiseptic property of iodine, since it was demonstrated by placing 1 drop of the standard 7 per cent solution of iodine in 3 ounces (85.02 Gm.) of culture medium; in this strength it inhibited the growth of bacteria. We also learned that 2 per cent iodine was successfully used as an irritant to the peritoneum by Johnson.[7] In view of these facts, we wonder, then, if the iodine in Pregl's solution did not exert some influence in counteracting the beneficial effects of the pepsin. With this in mind, pepsin without iodine was introduced. The method followed in extracting the pepsin required the use of an acid medium to effect as complete an extraction as was desirable. This need not be hydrochloric acid. We determined the hydrogen ion concentration by titration with the following results: pepsin extracted with hydrochloric acid gave a result of p_H 5.21 and pepsin extracted with glycerin, p_H 6.5. This showed that we had an acid extract. We further observed that pepsin extracted with glycerin digested blood clots in vitro.

Prior to the use of pepsin, we attempted to prove or discard the results of some experiments previously attempted. In the control experiment with rats the results after trauma with no agent introduced into the cavity were 100 per cent dense adhesions. We employed a 1 : 1,000 solution of urease and also obtained 100 per cent adhesions. Thiosinamine had been used by Offergell, Mitchell, Bush and Bibergeil with moderately good results. Our work with this drug showed that 58 per cent of the animals had from fine adhesions to none and that 16 per

10. Stewart, G. N.: Text Book of Physiology, ed. 8, New York, William Wood & Company, 1918.

11. Payr, E.: Ueber postoperative und spontane Adhaesionen in der Bauchhoehle, Zentralbl. f. Chir. **41**:99, 1914.

12. Ochsner, Alton: Personal communication to the authors.

cent of this number had no adhesions. Forty-two per cent had dense or moderate adhesions. This shows some measure of success. A proprietary preparation of bovine amniotic fluid was used and our results show 37 per cent dense or moderate and 63 per cent fine adhesions, somewhat more in keeping with the results of others, (Lacey [5]), but not as optimistic as the results of Trusler,[9] Johnson [7] and Warren.[8] Though our results showed 63 per cent of fine adhesions no incision was entirely free. These experiments were conducted on rats, and covered a total of 74 operations on 32 animals.

In our work with pepsin we used 44 animals, on which 104 laparotomies were performed. Most of the animals survived the several operations, but in those that died, we did not find a single case of intestinal obstruction at autopsy. Peritonitis and hemorrhage were the causes of death in all cases.

To produce adhesions which may be considered permanent, there must be sufficient trauma to produce an exudate capable of forming fibrin to a marked degree. Such a process may be set up by chemical irritation, by infection or by mechanical violence. In our series we determined by means of controls that the simple handling of the viscera resulted in the formation of adhesions in 6 of 8 cases, or 75 per cent, and when we traumatized with gauze we had adhesions in 8 of 10 cases, or 80 per cent. We therefore used the latter method.

The abdomen, from which the hair had been clipped close and to which iodine had been applied, was entered through a midline incision, and the small intestine was vigorously rubbed with a gauze-covered finger for a distance of from 6 to 8 inches (15 to 20 cm.) in several places. The peritoneum on each side of the

Adhesions Resulting from Operation on the Controls

	Number of Animals	Percentage of Adhesions			
		Class 1		Class 2	
		Number	Per Cent	Number	Per Cent
With trauma	10	2	20	8	80
Without trauma	8	2	25	6	75

incision was forcibly scraped with the sharp end of a scalpel. The contents of the abdomen were replaced and the abdomen was closed in layers, catgut being used for the peritoneum and dermal for the skin. Before the peritoneum was closed we instilled the fluid.[13] When the abdomen was reopened and required extensive separation of adhesions, we did not employ further trauma, except incident to separation.

For the purpose of classification we further subdivided the pathogenic adhesions into four types: (1) filmy (very fine cobweb strands, viscerovisceral or visceroparietal, easily separated); (2) fine (small strands of fibroconnective tissue uniting the viscera to the visceral or to the parietal peritoneum, easily separated); (3) moderate (the viscera adherent to the visceral or to the parietal peritoneum by contact or by bands requiring moderate force to separate them); (4) dense (fibrous adhesions between the viscera or between the viscera and the parietal peritoneum

13. The extracts of pepsin used in our experiments were prepared under the supervision of Dr. Sharp and Mr. Taylor of the Parke-Davis Laboratory. The active principle of pepsin was extracted from hog stomach with a 0.4 solution of hydrochloric acid and with glycerin and water. The strengths we used ranged from 0.5 to 2 per cent.

requiring considerable force or section tc separate, usually resulting in extensive trauma to the tissues involved and sometimes accompanied by bleeding).

We divided the cases into two classes: class 1, those in which we obtained filmy, fine or no adhesions, and class 2, those in which moderate or dense adhesions resulted. We used the hydrochloric acid extract in one series and the glycerin and water extract in another. The results follow:

Group 1 (hydrochloric acid).—Nineteen operations were performed; 45.5 per cent of the cases were in class 1, and of these 25 per cent showed no adhesions.

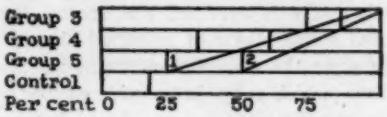

Fig. 1.—A composite graph showing prevention and alleviation curve in groups 3, 4 and 5 and in controls.

Fig. 2 (rabbit 28).—Third operation without injection eighteen days after lysis. The dense adhesion between the omentum, the small bowel and the peritoneum is notable.

Group 2 (glycerin and water).—Twenty-two operations were performed; 63.2 per cent of the cases were in class 1, and of these 39.1 per cent showed no adhesions. (Since the glycerin and water extract gave better results, we continued our experiments with this solution.)

Group 3 (glycerin and water, 0.5 per cent; traumatized by scarification).—Twenty-two operations were performed; 86.4 per cent of the cases were in class 1, and of these 75.8 per cent showed no adhesions.

Group 4 (glycerin and water, 0.5 per cent; lysis without further irritation).—Twenty-one operations were performed; 62 per cent of the cases were in class 1, and of these 33 per cent showed no adhesions.

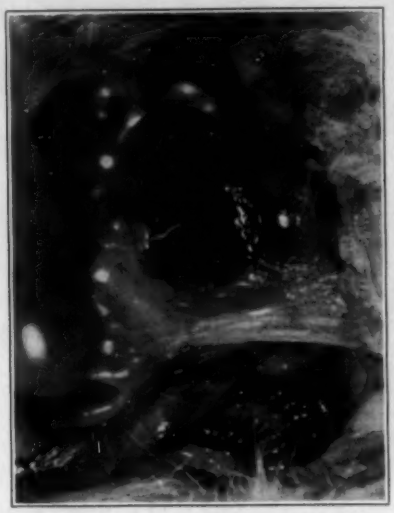

Fig. 3 (rabbit 15).—Third operation fourteen days following lysis; 50 cc. of 0.5 per cent pepsin in a solution of glycerin and water was injected. The adhesion of the appendix to the small bowel and to the peritoneum is apparent.

Fig. 4 (rabbit 19).—Fourth operation twenty-one days following lysis of a matted small bowel and an adhesion between the omentum and the appendix; 75 cc. of pepsin in a solution of glycerin and water was injected. There is one dense adhesion between two loops of the small bowel.

Fig. 5 (rabbit 35).—Twenty-two days after irritation and an injection of 50 cc. of 0.5 per cent of pepsin in a solution of glycerin and water. The one fine adhesion between the small bowel and the appendix can be seen.

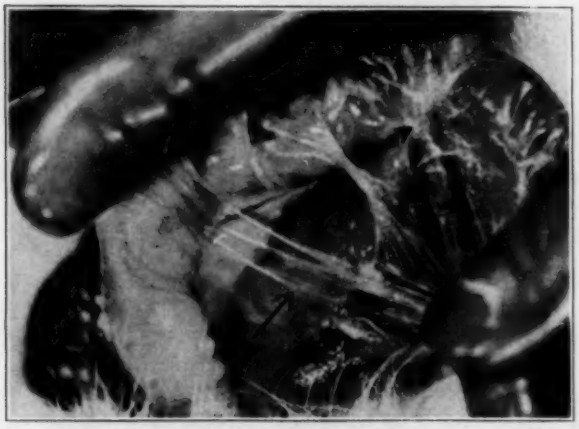

Fig. 6 (rabbit 38).—Operation thirteen days after a primary irritation (the small bowel was torn in the process and was repaired). Fifty cubic centimeters of pepsin in a solution of glycerin and water was injected. The repaired section of the bowel is adherent to the lower angle of the incision, and there are numerous cobweb adhesions.

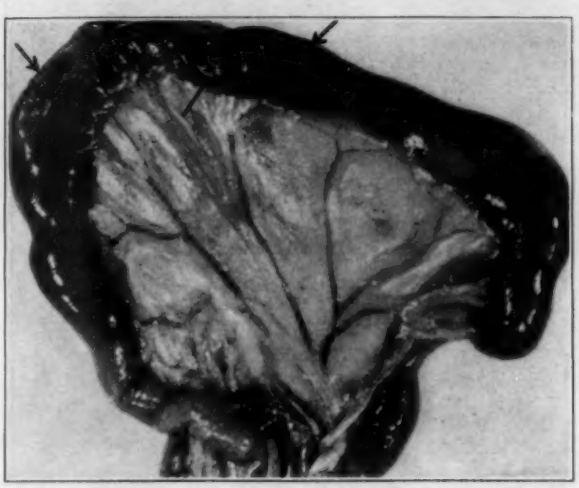

Fig. 7 (rabbit 37).—Operation twenty-four days after a primary operation, when the tissues were irritated. Fifty cubic centimeters of 0.5 per cent of pepsin in a solution of glycerin and water was injected. There are scars on the irritated section of the small bowel free from adhesions; edema of the mesentery is evident.

Fig. 8 (rabbit 27).—Operation thirty days after a primary irritation. Fifty cubic centimeters of 0.5 per cent of pepsin in a solution of glycerin and water was injected. The absence of adhesions is noticeable.

Fig. 9 (rabbit 32).—Operation twenty-one days after a primary operation. Twenty-five cubic centimeters of 0.5 per cent of pepsin in a solution of glycerin and water was injected. Note the peritoneal scar of the previous incision as well as the bowel free from adhesions.

Fig. 10 (rabbit 37).—Third operation eighteen days after irritation. Fifty cubic centimeters of 0.5 per cent of pepsin in a solution of glycerin and water was injected. Note the peritoneal scar of the previous incision and the absence of adhesions.

Group 5 (glycerin and water; lysis with irritation).—Twenty operations were performed; 50 per cent of the cases were in class 1, and of these 25 per cent showed no adhesions.

Since our results show the decreased tendency to the formation of adhesions we may assume that the benefit from the digestive action of the pepsin is realized before absorption takes place. There was a gradual decline in the incidence of adhesions with decreased trauma and in the presence of pepsin as follows:

Trauma Only	Class 1, per Cent	None, per Cent
Group 3	86.4	75.8
Group 4	62.0	33.0
Group 5	50.0	25.0

We see, then, that the greatest tendency in the reformation of adhesions is when trauma is induced in the presence of existing adhesions and their separation; and we therefore realize the futility of any effort to prevent the reformation of adhesions in cases in which additional trauma is induced.

COMMENT

It is apparent from our results that there is some hope in the solution of the problem of the formation of adhesions, with the aid of a solution of pepsin, since this substance meets with common requirements in our experimental work. Recovery was prompt and the adhesions were considerably lessened.

In the study of the prevention of adhesions one must bear in mind that it is not desired to eliminate or prevent all adhesions. Certain adhesions are not only desirable, but necessary. The proper perspective must not be lost, for the problem is one of preventing the formation or the reformation of only such adhesions as are detrimental to postoperative convalescence and to future health.

It would not be amiss to mention briefly the prophylactic measures for the prevention of adhesions as outlined by Payr: (1) strict asepsis; (2) gentleness in handling the tissues; (3) elimination of sponging and draining when possible; (4) exact hemostasis; (5) careful packing off of the intestines, but avoidance of unnecessary packing; (6) limitation to as little cutting as possible (this does not apply to preexisting adhesions; sharp dissection of these, when lysis is necessary, is preferable to blunt dissection); (7) peritonealization of every ligature stump; (8) careful aspiration of the peritoneal fluid. Satisfactory results obtained by the application of Payr's measures justify the additional time consumed during an operation. As an addition to the exercise of these principles one may employ substances to alleviate a condition which is inevitable in from 15 to 20 per cent of all operations.

When an infection is present, and when adhesions are desirable, the efforts at limitation will not be delayed by any favorable solution or

foreign substance introduced into the cavity. The factors active during such times are very definite. The close approximation of inflamed surfaces results in delayed absorption of fibrin, which is not subject to attack as on exposed surfaces. We do not see how a substance injected would interfere with the formation of physiologic adhesions when infection is present and when adhesions are necessary. The formation of adhesions is dependent on deposits of fibrin and the quantity of fibrin deposited at any given point is in turn dependent on several factors, among which are: (1) the extent of the trauma, and (2) the extent of the infection. Though cases are found in which considerable trauma was used or in which infection was present, but in which no adhesions were found later, these cases were the exception and not the rule.

We think it can be assumed that the rôle of fibrin, when it is required, is not destroyed by foreign substances and that when adhesions are desirable fibrin is present in sufficient amount to ward off the invasion of a foreign substance.

SUMMARY

Attempts have been made for forty-five years to introduce methods for the prevention of adhesions, but all have been discarded. The benefit of digestive ferments has not been disproved. Thiosinamine has not been effective in our experiments. A proprietary preparation of bovine amniotic fluid gave favorable results. Pepsin, as a digestant, has been suggested experimentally to be an adjunct to the general principles of good surgery in the prevention of adhesions. Pepsin extracted with hydrochloric acid was not promising as a preventive, but in a glycerin and water extract it was effective in its prevention or reduction of the reformation of adhesions in 62 per cent of the cases. Pepsin was most effective as a prophylactic in primary operations in 86.4 per cent of the cases.

PROGRESSIVE OBSTRUCTIVE JAUNDICE

CHANGES IN CERTAIN ELEMENTS OF THE BLOOD AND THEIR RELATION TO COAGULATION

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AND
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In 1618, according to Thudichum,[1] Johannes Fabricius removed gallstones from the gallbladder of a living subject. Fabricius Hildauus refers to this operation in his "Surgical Observations," but it is not clear whether the operation was premortem, as the only evidence of the subject's being alive is the somewhat dubious expression "delineatio horum lapidum ad vivum facta."

No such shadow hangs over Petit's [2] first project. During the decade beginning with 1733 he discussed boldly and with great acumen tumors of the gallbladder and the sequences of biliary obstruction. From these papers and the discussion which arose over them he emerged as the founder of gallbladder surgery, his description of symptoms and outlines of operative procedures remaining classic.

Neither Petit nor Thudichum, who wrote with equal insight a century and half later, failed to evaluate the difficulties of gallbladder surgery excepting one. Neither mentioned the hemorrhagic tendency in obstructive jaundice which today stands as one of the most difficult and perplexing problems confronting the modern physician. Nor did Bobbs [3] of Indianapolis, who fathered the modern cholecystotomy, or Langenbuch [4] in 1882, who is credited with the first cholecystectomy, refer to the dangers of postoperative hemorrhage although their statistics would have been better and the results of Thiriar and Courvoisier after them would have been more successful had they known of the danger of hemorrhage and of a way to control it.

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This work has been conducted under grants from the Christine Breon Fund for Medical Research and from the Research Budget of the Department of Pathology of the University of California Medical School.

1. Thudichum, J. L. W., quoted by Smith.[5]
2. Petit, J. L.: Mém. Acad. roy. de chir. **1**:163, 1743.
3. Bobbs, J. S.: Tr. Indiana State M. Soc., 1868, p. 68.
4. Langenbuch, quoted by Smith.[5]

The modern surgeon stands superior to these venerable pioneers in technical knowledge and skill and knows of the danger from hemorrhage since Greig Smith [5] remarked on it forty years ago, but in spite of an enormous investigative effort and the propagation of innumerable theories he knows no more of the reason for, or of the method to control, this bleeding than did Martin Sims [6] or Petit or Johannes Fabricius before him.

All the theories as to the cause and the treatments based on them to date have concerned themselves with deficiencies in one or another of the formed clotting elements of the blood in spite of the fact that various observers have at different times found each of these elements to be normal, even in extreme examples. It is true that Howell [7] proposed a deficiency in heparin as the possible cause of bleeding, but aside from this one instance investigators have believed and attempted to prove that a variation in one or more of the formed clotting elements is responsible for death due to hemorrhage of these unfortunate persons.

The first of these theories apparently developed from the work of Rohig,[8] who believed that the circulating bile salts (taurocholate and glycocholate) were responsible for the bradycardia and lowered blood pressure. King and Stewart [9] repeated his work in 1909 and in addition to duplicating his result discovered that bile pigments in combination with the simple salts of calcium were less toxic than when given alone. They concluded that the reserve blood calcium was used up in combining with excess bile pigments and therefore was not available for coagulation.

Many years later W. Walters [10] published the results of autopsies on thirty jaundiced patients who died after abdominal operation. Half of these had large intraperitoneal hemorrhages which he accounted for by assuming a deficiency in calcium. He cited the work of Lee and Vincent,[11] who found that the addition of calcium chloride to jaundiced blood in the test tube shortens the coagulation time. Reasoning from this he advocated the use of calcium chloride intravenously in jaundiced patients. This medication met with some success and is still being used.

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5. Smith, Greig: Abdominal Surgery, Philadelphia, P. Blakiston's Son & Co., 1891, p. 610.

6. Sims, Martin: Brit. M. J. **1**:811, 1878.

7. Howell, W. H.: Am. J. Physiol. **77**:680 (Aug.) 1926.

8. Rohig, J.: Arch. d. Heilk. **4**:385, 1863.

9. King, J. H., and Stewart, H. A.: J. Exper. Med. **11**:673, 1909. King, J. H.; Bigelow, J. E., and Pearce, L.: J. Exper. Med. **14**:159, 1911.

10. Walters, Waltman: Surg., Gynec. & Obst. **33**:651 (Dec.) 1921.

11. Lee, R. I., and Vincent, B.: The Relation of Calcium to the Delayed Coagulation of the Blood in Obstructive Jaundice, Arch. Int. Med. **16**:59 (July) 1915.

The benefit, however, is not due to replacement of deficient blood calcium but to an entirely different mechanism which will be discussed later.

Immediately following publication of the theory on calcium deficiency many workers made determinations of blood calcium on jaundiced patients and on experimental animals. Halverson, Mohler and Bergeim [12] reported a slight reduction in the serum calcium in jaundice, but concluded that the reduction was more apparent than real. Koechig [13] confirmed this work. Walters and Bowler,[14] Snell, Greene and Rowntree,[15] and Zimmerman [16] found no change in the serum calcium, either in obstructive or in other forms of jaundice.

Following the development of methods for estimating the diffusible fraction of the serum calcium it was felt that perhaps the deficiency of this substance accounted for the bleeding. Kirk and King [17] reported moderate reductions in the total calcium with a much greater decrease in the diffusible fraction. They emphasized the importance of diffusible calcium in the clotting complex. Emerson [18] corroborated their observations and drew attention to possible errors due to ether anesthesia. Buchbinder and Kern,[19] on the other hand, found no alteration in the serum calcium after ligation of the common duct in adult dogs, although they did find a progressive decrease of this substance in puppies. Gunther and Greenberg [20] substantiated this work and stated that there was no proof of a deficiency of available calcium in the blood serum of jaundiced patients. It is true that they found the concentration of nondiffusible calcium low in a few instances, but this was accounted for by a lowered concentration of serum albumin. They materially advanced the entire subject by concluding that factors other than a deficiency in calcium must be sought as an explanation of the tendency to bleeding in obstructive jaundice.

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12. Halverson, J. O.; Mohler, H. K., and Bergeim, O.: Calcium in the Blood in Tuberculosis, J. A. M. A. **68**:1309 (May 5) 1917.

13. Koechig, I.: J. Lab. & Clin. Med. **9**:679 (July) 1924.

14. Walters, W., and Bowler, J. P.: Surg., Gynec. & Obst. **39**:200 (Aug.) 1924.

15. Snell, A. M.; Greene, C. H., and Rowntree, L. G.: Disease of the Liver: II. A Comparative Study of Certain Tests for Hepatic Function in Experimental Obstructive Jaundice, Arch. Int. Med. **36**:273 (Aug.) 1925.

16. Zimmerman, L. M.: Am. J. M. Sc. **174**:379 (Sept.) 1927.

17. Kirk, P. L., and King, C. G.: J. Lab. & Clin. Med. **11**:928 (July) 1926.

18. Emerson, W. C.: J. Lab. & Clin. Med. **14**:122 (Nov.) 1928.

19. Buchbinder, W. C., and Kern, R.: Experimental Obstructive Jaundice: II. Modification of the Parathyroid Tetany Mechanism in Jaundice, Arch. Int. Med. **41**:754 (May) 1928.

20. Gunther, L., and Greenberg, D. M.: I. The Diffusible Calcium and the Proteins of the Blood Serum in Jaundice, Arch. Int. Med. **45**:983 (June) 1930.

With the theory of calcium deficiency excluded, the attention of workers was directed toward possible changes in fibrinogen. The suspicion was reasonable, for Howe,[21] Mann and Bollman,[22] Foster and Whipple,[23] and others had shown the liver to be the main source and possibly the only source of fibrinogen, and obstructive jaundice with subsequent damage to the liver could be expected to cause a reduction in the total amount of fibrinogen. On this basis, Full [24] and McLester [25] concluded that the fibrinogen content of the plasma might be used as an index of hepatic function, but Peters and Van Slyke [26] showed that no parallel exists between fibrinogen and the degree of hepatic function, unless the damage to the liver is far more severe than that seen in obstructive jaundice.

Foster and Whipple agreed in finding an increase in fibrinogen following inflammatory and destructive hepatic lesions unless the loss of liver substance was so great that the organ could no longer meet the demands of the body, when a terminal drop occurred. Studies made on the blood fibrinogen in various conditions showed wide variations which were not entirely understood until Wiener and Wiener [27] reported the physiologic limits of the concentration of fibrinogen to be extremely broad. They concluded that any irritation or stimulation of the liver raised the level for fibrinogen, and they found it high during menstruation and pregnancy, following infections, exposure to roentgen ray and ingestion of chloroform and phosphorus, and in early cirrhosis. Gram [28] reported four cases of extreme jaundice from malignant obstruction with extremely high fibrinogen levels.

From a study of the literature we have found that the total fibrinogen increases consistently wherever there has been damage to the tissue, including pregnancy, hemorrhage and infection, with a general rise in malignancy excepting when the liver is radically replaced by metastases.

Moss [29] concluded that in obstructive jaundice there was no deficiency but rather an increase in fibrinogen relative to the extent of damage to the liver. With these results in hand he was forced to con-

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21. Howe, P. E.: J. Biol. Chem. **57**:235 (Aug.) 1923.

22. Mann, F. C., and Bollman, J. L.: Proc. Staff Meet., Mayo Clin. **4**:328 (Nov.) 1929.

23. Foster, D. P., and Whipple, G. H.: Am. J. Physiol. **58**:407, 1922.

24. Full, H.: Verhandl. d. Kong. f. innere Med. **33**:201, 1921.

25. McLester, J. S.: The Diagnostic Value of Blood Fibrin Determinations, J. A. M. A. **79**:17 (July 1) 1922.

26. Peters, J. T., and Van Slyke, D. D.: Quantitative Chemistry, Baltimore, Williams & Wilkins Company, 1931.

27. Wiener, H. J., and Wiener, R. E.: Plasma Proteins, Arch. Int. Med. **46**: 236 (Aug.) 1930.

28. Gram, H. C.: Acta med. Scandinav. **56**:107, 1922.

29. Moss, Walter: Experimental Obstructive Jaundice; Its Effect on Fibrinogen and Coagulation of the Blood, Arch. Surg. **26**:1 (Jan.) 1933.

clude that hemorrhage in jaundice was not due to or associated with a deficiency of fibrinogen in the plasma.

Of other clotting components little can be said. The platelets have repeatedly been shown to be within normal limits in these cases. The methods of estimation of thrombin are inaccurate, and although we have extracted thrombin from the clots of jaundiced subjects, there is no quantitative estimation worthy of the name. Howell's [30] claim that a variation in the amount of heparin is the probable cause of bleeding in hemophilia and jaundice is just as uncertain, as there is as yet no quantitative method of determination for heparin.

The importance of dextrose in the clotting complex has been questioned. Partos and Svec [31] showed that substances which increased the coagulability of the blood mobilized glycogen and produced hyperglycemia while substances which prolonged the coagulation time caused hypoglycemia.

Cannon and Gray [32] found that epinephrine increased both the blood sugar and coagulability. Cowan and Wright [33] suggested that this is due to the influence of dextrose in raising the blood calcium. We have found that dextrose will favorably affect persons with jaundice who tend to bleed and that it exercises a protective effect when given preoperatively. The reasons, still obscure, will be discussed later.

Methods for the prognosis of bleeding in obstructive jaundice have been as unsatisfactory as a study of the reasons for bleeding. Ravdin, Riegel and Morrison [34] have agreed with Bancroft, Kugelmass and Stanley-Brown,[35] that coagulation and bleeding times are of no value in determining a tendency to bleeding, and we have found a normal or even shortened coagulation time in the face of fatal hemorrhage.

EXPERIMENTAL WORK

Because of the voluminous contradictory literature and previous isolation of various phases of the subject into studies of single elements, we decided to approach the subject of bleeding in obstructive jaundice by making all possible estimations of the formed clotting elements of the blood on the same patients at the same time, and thus experimentally compile the evidence which had previously been accumulated from the literature.

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30. Howell, W. H.: Am. J. Physiol. **71**:553 (Feb.) 1925; Bull. Johns Hopkins Hosp. **42**:199 (April) 1928.

31. Partos, A., and Svec, F.: Arch. f. d. ges. Physiol. **218**:209, 1927.

32. Cannon, W. B., and Gray, Horace: Am. J. Physiol. **34**:232, 1914.

33. Cowan, P. W., and Wright, H. N.: Am. J. Physiol. **100**:40, 1932.

34. Ravdin, I. S.; Riegel, C., and Morrison, J. L.: Ann. Surg. **91**:801 (June) 1930.

35. Bancroft, F. W.; Kugelmass, I. N., and Stanley-Brown, M.: Ann. Surg. **91**:161, 1929.

Fourteen patients in the surgical wards were selected for this study. They all had experienced prolonged obstructive jaundice which, with two exceptions, was due either to stones or to tumors. There was one case of biliary cirrhosis and one of arsphenamine poisoning. A few patients had superimposed infections such as cholecystitis and cholangeitis. Thirty-five cubic centimeters of blood was taken from each patient, and the coagulation time, icterus index, red cell volume, sedimentation time, fractions for total and diffusible serum calcium and values for fibrin and fibrinogen were determined.

We have regarded the icterus index as the index of progression corresponding roughly to the severity and duration of the disease in these cases. The coagulation time was determined in an 8 mm. tube, after the method of Lee and White.[36] Sedimentation time was determined by placing 0.8 cc. of blood in 0.2 cc. of a 3.8 per cent solution of sodium citrate and recording the time required for the cells to sediment 18 mm.

The determination of fibrinogen was made after the method of Samson[37] with certain modifications. The fibrinogen in citrated plasma was precipitated by the addition of an equal volume of saturated solution of sodium chloride, then packed firmly at the bottom by centrifugation for thirty minutes at 3,000 revolutions per minute, and read directly from calibrated markings on the wall of the tube. While there is some possibility of including other plasma proteins by this method, the fractions, according to Pickering,[38] are thrown down rather independently, and for comparative use the method is quite satisfactory.

The fibrin was coagulated from citrated plasma, as recommended by Gram,[28] by the addition of dilute solutions of calcium chloride and sodium chloride, after which it was thoroughly dehydrated and weighed. Two cubic centimeters of plasma were used for each fibrin determination.

Calcium was determined, after the manner of Gunther and Greenberg,[20] by titration with potassium permanganate. The diffusible fraction was obtained by drawing it through a semipermeable collodion membrane by water suction.

The table shows the results of these determinations on the fourteen patients, tabulated in order from the lowest to the highest icterus index.

Results of Determinations of Blood Elements on Fourteen Patients

Number	Icterus Index	Clotting Time in Minutes	Sedimentation Time in Minutes	Diffusible Calcium	Total Calcium	Fibrinogen, Cc. per 5 Cc. Plasma	Fibrin, Gm. per 2 Cc. Plasma
1	2.0	9.0	360	4.600	....	0.30	0.0320
2	15.0	4.5	45	.....	9.24	0.60	0.0600
3	17.0	3.5	75	3.022	18.60	0.35	0.0900
4	18.6	4.5	240	7.200	....	0.30	0.0300
5	30.0	7.0	30	4.640	6.33	0.49	0.0429
6	33.0	6.0	15	3.550	8.32	0.60	0.0690
7	40.0	11.0	40	.....	6.56	0.70	0.0730
8	45.0	11.0	12	4.350	6.73	0.50	0.0649
9	50.0	17.0	35	.....	6.10	0.75	0.0700
10	66.0	2.5	20	3.810	....	0.60	0.0460
11	100.0	25.0	28	2.120	8.45	0.75	0.1110
12	104.0	9.0	28	4.370	8.72	0.55	0.0500
13	143.0	9.0	360	5.280	....	0.15	0.1110
14	150.0	9.0	40	4.640	8.45	0.25	0.1200

36. Lee, R. I., and White, P. D.: Am. J. M. Sc. **145**:495, 1913.

37. Samson, K.: Med. Klin. **24**:217 (Feb. 10) 1928.

38. Pickering, J. W.: The Blood Plasma in Health and Disease, New York, The Macmillan Company, 1928.

The coagulation time was normal for the majority of the patients, although for one it was twenty-five minutes. He bled for three days from a paracentesis abdominis needle wound. The next highest values were twelve and one-half minutes in one case and eleven minutes in two cases. The patient with the twenty-five minute clotting time had a carcinoma of the common duct with ascites. He had been jaundiced for six weeks and had an icterus index of 100. All of the clotting elements were within normal limits.

Values for diffusible calcium were low throughout the group because of the titration method used. Values for total calcium were low except for the patients who were receiving calcium chloride intravenously.

The fibrinogen level increased with the onset of jaundice. The patients with an index of from 50 to 100 who had been jaundiced for from three to six weeks had the highest values. The extremely jaundiced patients with indexes above 100 had values back to normal or below. Relative to this, Schmidt and Brakefield [39] found that bile pigments and acids are excreted in the urine of dogs in maximum amounts approximately two weeks after ligation of the common duct and that from then on the amounts gradually diminish. They attribute this to a decrease in synthesis. Snell, Greene and Rowntree [15] also found an increase of bile acids in the blood to a certain point, then a gradual decrease.

The fibrinogen, especially in the jaundiced patients revealing high values, appeared as a more finely divided precipitate than that in normal blood as though the small particles did not adhere to each other. The values for fibrin varied somewhat but did not show a characteristic change in amount. We did notice, however, that in the cases of deep jaundice the fibrin clots did not retract. They were large, contained a great deal of fluid and were deeply stained. They were less elastic and more easily torn apart than those from normal blood.

ANIMAL EXPERIMENTATION

While these observations on patients substantially corroborated the general opinion in the literature, we felt that results would be far more conclusive if they were checked against similar determinations made on carefully controlled laboratory animals.

Dogs were chosen for this purpose as they do well on the average human diet, are large enough to offer little surgical difficulty and have an extrahepatic biliary system quite similar to the human structure. Six animals of average size and mixed breeds were used. After a quarantine period of two weeks, the common

39. Schmidt, C. L. A., and Brakefield, C. L.: J. Biol. Chem. **67**:523 (Feb.) 1926.

bile ducts were severed between double silk sutures near the duodenum. A few accessory ducts were seen and ligated, and on two occasions it was necessary to operate a second time to secure accessory ducts missed at the first entry. Subsequent autopsies showed no ducts which had not been surgically treated.

Excluding one rather old animal which died of pneumonia during the third week, the dogs lived, on an average, for three and a half months after ligation. They went through an initial stage of lethargy and anorexia lasting approximately two weeks. Then they began to take a balanced diet of meat (cooked and raw), milk, rice, potatoes, carrots and bread in generous amounts. They became normally active, taking daily exercise in the open air pens and sleeping in cages in steam-heated rooms at night. They lost weight gradually until about the beginning of

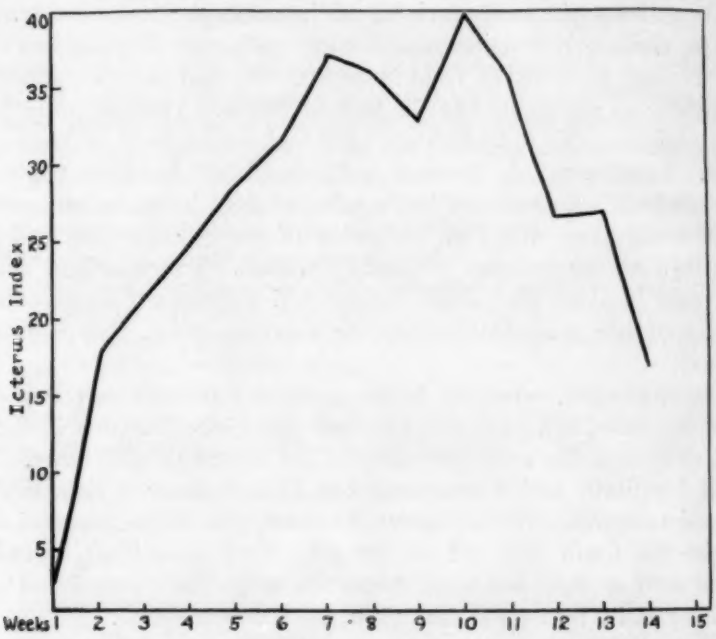

Fig. 1.—Changes in the icterus index in five dogs following ligation of all the excretory bile ducts (composite curve).

the third month, when the loss in weight was accelerated. They again became lethargic and ate little or nothing. Two of the five dogs died of spontaneous hemorrhage into the bowel. One had bleeding into the peritoneal cavity from the liver in the region of the gallbladder. His coagulation time was four and one-half minutes on the day before death. The remaining two apparently died of biliary cirrhosis, characterized by the typical liver, ascites and collateral circulation. Blood was taken from these animals before operation, and determinations were made of the icterus index, coagulation time, sedimentation time, fibrinogen, fibrin, red cell volume, diffusible calcium and total calcium in the manner heretofore described. After operation these observations were made at regular weekly intervals until death (fig. 1). The curves published here are composite averages of the entire group. They coincide with the individual graphs, which are omitted to save space.

The icterus indexes increased rapidly after operation until about the seventh week, when they either became stationary or decreased slightly. Terminally they showed a tendency to decrease rapidly. The stationary period may represent a phase of decreased synthesis or a stage of maximum storage of bile. If a recirculation of bile pigment existed, it is possible that the added synthetic burden placed on the reticulo-endothelial system reached a peak at this time. The final drop occurred for no valid reason, except perhaps a marked terminal decrease in the food intake of the animals.

Hematocrit readings showed an initial drop, presumably from the blood lost at operation, but possibly the change in osmotic pressure of the plasma decreases the individual cell volume (fig. 2). They then remained constant until a final drop occurred from hemorrhage just

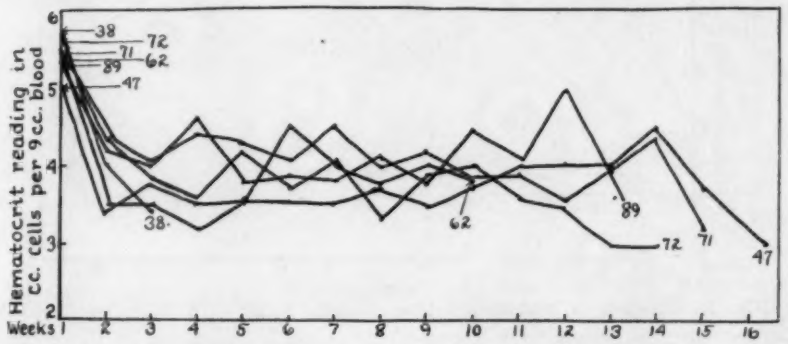

Fig. 2.—Hematocrit readings in five dogs following ligation of the common bile duct.

before death. The animals became dehydrated in the terminal stages, and these constant figures must indicate a loss of red cells, although accurate studies of the blood will have to be reported later.

The coagulation time was not increased beyond normal in any case, but there was a rather constant and significant shortening shortly before death (fig. 3). This terminal change was particularly significant, as will be shown later.

The fibrin revealed no characteristic change in weight, as shown by the curve in figure 4. In the last third of the experimental period, the masses of fibrin were much larger, although they weighed no more after drying. This failure of retraction was constant whether the clots were dried or left in the serum where they formed.

The fibrinogen, although not markedly changed in amount, showed the same inverse ratio to sedimentation as was found in the group of patients. There was an initial rise and then a drop to slightly below normal, where the fibrinogen remained constant (fig. 5 *A*).

Each specimen of fibrinogen was examined for elasticity and cohesive power by teasing it out on filter paper with glass rods. In the beginning it was an elastic rubbery mass which stretched into thin strands on being torn apart. As time elapsed after the operation, the fibrinogen was observed to sediment much more slowly in the tube; the particles appeared smaller, and after centrifugation there was no elasticity to the mass; it flattened out on filter paper from its own weight and was

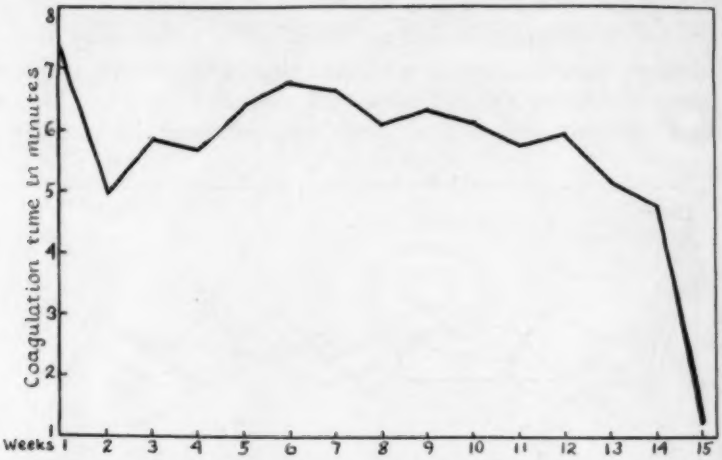

Fig. 3.—Changes in coagulation time in dogs in series 1 (composite curve). The excretory bile ducts were ligated.

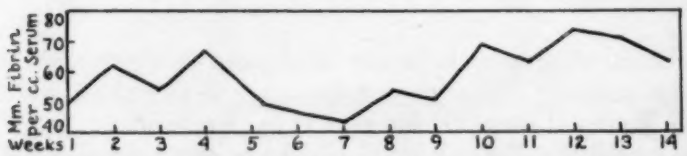

Fig. 4.—Determinations of blood fibrin on five dogs in series 1 with obstructive jaundice (composite curve).

easily torn apart. This change in the consistency of fibrinogen closely followed the nontractility and changes in consistency of the formed fibrin mesh previously described.

Sedimentation time showed an initial rapid drop as low as twelve minutes, then a gradual recovery to about three fourths of the normal time and then a final drop before death (fig. 5 *B*). The curves in figure 5 were obtained by plotting sedimentation time against elapsed time. This corresponds to the curve obtained for the group of patients and varies inversely with the amount of fibrinogen.

Values for total and diffusible calcium remained fairly constant throughout the experiment (fig. 6). There was no characteristic change in them with the progress of jaundice.

The results of the observations of other writers and our own experience with patients in the hospital and with groups of experimental animals, following ligation of the common duct, show that there is no significant variation in the amounts of the formed clotting elements of the blood; the only important change is the loss of retractile power of the fibrin and whole clot, a shortening of coagulation time shortly

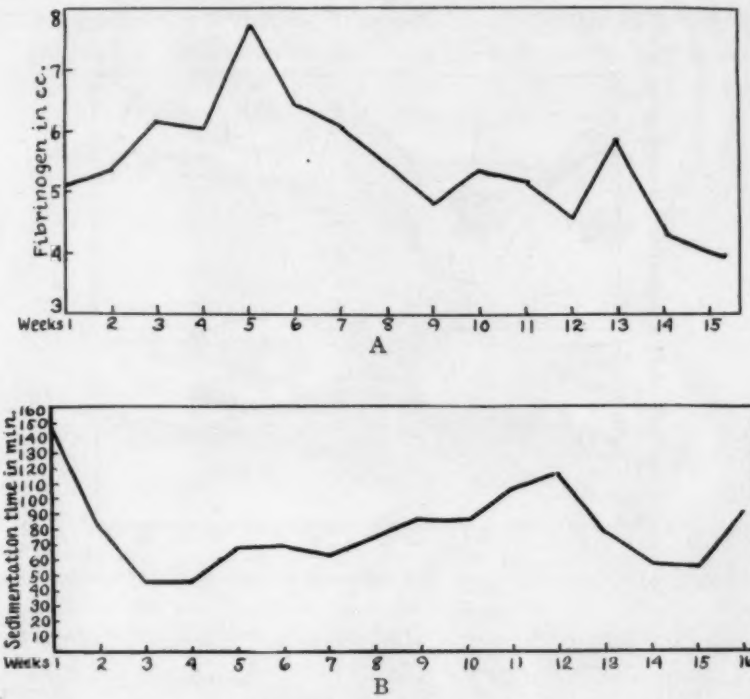

Fig. 5.—*A*, changes in blood fibrinogen in animals of series 1 following ligation of the excretory bile ducts (composite curve); *B*, changes in sedimentation time in five dogs after ligation of the excretory bile ducts (composite curve). Note the striking inverse proportion of the curve in *A* as compared to *B*.

before death and the imperfect precipitation of an altered fibrinogen in the plasma of these bloods. With these facts it becomes apparent that the error in clotting is due to an additional product which inhibits the process or interferes with the clotting mechanism rather than to an insufficient amount of any of the elements contributing to the normal clotting complex.

Mann has shown that dogs that have undergone hepatectomy die with hypoglycemia. That this is not the only factor in their death,

however, is indicated by the behavior of such animals when given large quantities of dextrose. Instead of dying in the usual time, they manifest a second syndrome closely similar to that of the late stages of obstructive jaundice with jaundice, convulsions, hemorrhage and coma, presumably due to the accumulation in the plasma of split protein products which the liver would normally remove. As cysteine and other mercaptans are excreted by carnivores as taurine in the bile, as well as broken to urea in the liver and converted by the liver to dextrose in small amounts, it was felt that taurine, cysteine and related products are the protein products which most probably back up in the blood and collect in sufficient concentration in the circulating plasma to cause changes in the

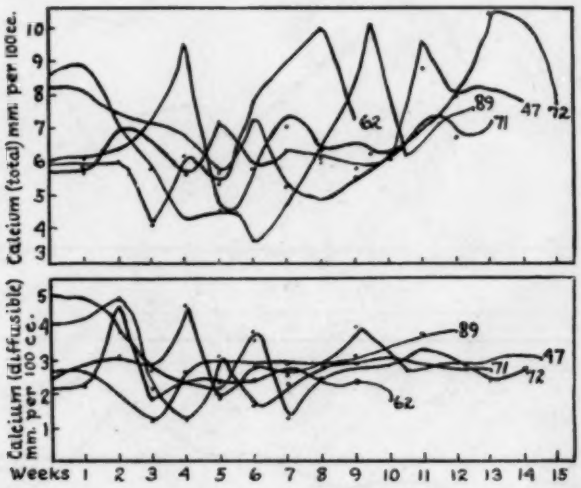

Fig. 6.—Total serum calcium and diffusible calcium in dogs in series 1.

clotting mechanism. Cysteine, glutathione and polyanethylthione have been reported as inhibiting the clotting of blood. Denole and Remert [40] have produced multiple hemorrhages in rabbits by the intravenous injection of polyanethylthione. Mueller and Sturgis [41] have observed that cysteine is an anticoagulant. They placed 0.1 Gm. each of cysteine and of alanine—which is identical except for the mercaptan (— SH) radical—in 1 cc. of blood. The blood containing alanine coagulated in ten minutes or less (normal), while that containing cysteine did not clot in twenty-four hours.

We repeated the experiment, using graduated higher dilutions of cysteine up to one containing 0.001 Gm. When present in amounts sufficiently

40. Denole, V., and Remert, M.: Arch. f. exper. Path. u. Pharmakol. **158**:211, 1930.

41. Mueller, J. H., and Sturgis, S.: Science **75**:140 (Jan. 29) 1932.

minute to allow clots to form, cysteine shortens the coagulation time and changes the normal clot to a dark, friable, nonretractile mass closely resembling the clot formed by the blood of jaundiced dogs shortly before they die of spontaneous hemorrhage. Calcium added to the plasma, both in vitro and circulating, temporarily prevents this, while cysteine added to precipitated fibrinogen causes it to swell, forming globoid particles which float. Figure 7 shows the increasing retractile power of the clot as the concentration of cysteine is decreased, the figures under the tubes representing volumes of 10 per cent solution of cysteine hydrochloride.

These facts suggest that cysteine influences blood clotting, affecting both the speed of clot formation and the consistency of the product.

It has been shown that cystine injected into the peripheral circulation can be recovered from other veins shortly afterward in small

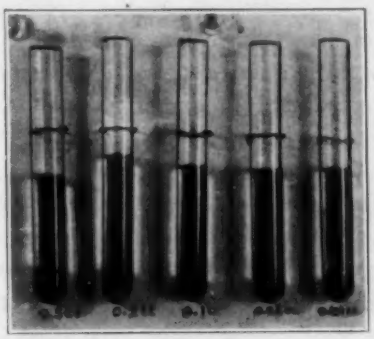

Fig. 7.—Changes in the size of the clot with graduated dilutions of cysteine. Each tube contains 1 cc. of blood. The clot on the left is the largest. It forms almost instantly and is so friable as to flow on tilting the tube. The clot on the extreme right is the most retractile, therefore the smallest, and forms in approximately four minutes. However, it is definitely more friable and forms more rapidly than in unadulterated blood, even though such a minute quantity as 0.001 Gm. of cysteine has been added. The addition of 1 cc. of 2 per cent solution of calcium chloride to each tube prevents these phenomena. When the same dilution of cysteine is produced in vivo (calculating blood volume from body weight) in a normal animal, blood withdrawn within ten minutes exhibits a decreased coagulation time and a friable clot.

amounts, but if it is placed in the mesenteric vein none can be recovered, the liver apparently removing it from circulation by a reduction of cystine to cysteine, then through several intermediate compounds such as glutathione (with one molecule of glutamic acid) and polyanethylthione. That this is not a simple reaction in the plasma but rather an organic function of the liver is evident because cysteine added to whole blood in the test tube may be recovered many hours afterward.

The normal synthesis of taurine, which is excreted in large amounts by carnivores and man, is thought to start with cysteine. Sulphur, sulphates and thiosulphates have no appreciable effect either on the speed of clotting or on the type of clot formed, the mercaptan radical apparently being the essential influence.

In animals with acute necrosis of the liver after poisoning with carbon tetrachloride the neutral sulphur fraction (which included the mercaptans) of the urine increases. Cystine, tyrosine and leucine are excreted in the urine. Guanidine, with this specific necrosis, is one of the split protein products which apparently collects in excess in the circulation. While it inhibits clotting it does not cause the formation of a poor clot with nonretraction in small amounts, as does cysteine.

If the metabolism and excretion of proteins containing this radical are functions of the liver, the intermediate products, including the mercaptans, must also accumulate in the circulation when excretion through the liver is obstructed by common duct block. From their effects on coagulation in vitro it seems reasonable that they would exercise a similar effect when collected in excessive amounts in the circulating plasma.

Normal animals when given larger doses of cysteine hydrochloride intravenously show the same changes in clotting as those with a long-standing biliary obstruction. The clotting time diminishes, and the fibrin mass is spongy and friable and does not retract.

The food source of these products is chiefly meat, and dogs and other carnivores excrete large amounts of taurine in the bile. Human bile contains taurine in moderate amounts, while the excretion from the liver of rabbits, sheep and herbivores generally contains little of this or allied substances.

With the postoperative behavior of dogs with common duct block under normal conditions tentatively established by the group of six animals described, a second group of four was selected, and the common ducts were ligated. To establish the optimum dietary conditions for the production of mercaptan and cysteine, these animals were put on a diet consisting solely of animal protein. The average postoperative survival period of the group was five and a half weeks as compared with thirteen weeks in the group receiving a balanced ration. All of the animals bled badly during the terminal stages of life, the large bowel of each being filled with blood at autopsy. A control animal thrived during this time on the same diet and under the same conditions. Samples of venous blood were taken each week, being collected in sodium citrate under liquid petrolatum which contained no sulphur. In

each of these the Fleming [42] test for cysteine became positive after jaundice had developed and was more strongly positive as the illness progressed, although after the middle of the period the icterus index declined. The Fleming test was not well suited to plasma, however, and after an interval of use Grote's [43] method was substituted and found more applicable. The end-results remained the same. The Grote test was accomplished by withdrawing 1 cc. of plasma from beneath the oil, placing it in a test tube with an excess of powdered sodium bicarbonate and immediately layering 1 cc. of the reagent on it. After fifteen minutes a thin band of violet appears at the junction of the fluids and immediately below a band of green. These bands exhibit a maximum intensity after about one hour, then gradually fade and disappear entirely in twenty-four hours. We divided the results into four groups, negative, 1+, 2+ and 3+, depending on the intensity and on the width of the bands. Normal plasma, as well as plasma to which small quantities of bile has been added, produces a negative reaction. In from a week to ten days after ligation of the common duct, the plasma of a dog on a meat diet becomes 1+. In approximately three weeks it is 2+, and when the tendency to bleeding is fully developed it is usually 3+.

While experimentally applying other color tests, we tried therapeutic measures to establish cysteine as an anticoagulant in obstructive jaundice. Clinically, calcium has proved of some value, as it apparently improves the consistency of the clot and has been reported to raise the blood sugar. The intravenous administration of large quantities of dextrose has also been used with some success, and is reported to have raised the blood calcium. This mechanism is not fully understood, but it is known that cysteine may be partially changed to dextrose with the elimination of sulphates and sulphites. Calcium may influence this reaction. Sodium plumbite will convert cysteine to disulphides, but its use is obviously dangerous. Magnesium oxide also breaks the cysteine molecule, causing the formation of an insoluble salt. Ultraviolet rays have been thought to influence cysteine catabolism. We finally selected monobrombenzene as the most useful substance, as it is relatively nontoxic, can be given either orally or intramuscularly, and combines with cysteine to form only mildly toxic bromphenylmercapturic acid and ethereal sulphates which are excreted by the kidneys and which may be isolated from the urine (fig. 8).

Abderhalden and Wertheimer [44] have found that animals given brombenzene excrete moderate amounts of bromphenylmercapturic acid

42. Fleming, R.: Biochem. J. **24**:965, 1930.

43. Grote, I. W.: J. Biol. Chem. **93**:25, 1931.

44. Abderhalden, E., and Wertheimer, E.: Ztschr. f. physiol. Chem. **201**:267, 1931.

when on a pure meat diet, a little of this acid when starved, and practically none when given carbohydrates. Bromphenylmercapturic acid appears in the urine as bundles of needle-like crystals, and the number isolated is a partial index of the relative amounts of cysteine and brombenzene in the circulating blood. The remaining fraction, however, is eliminated as ethereal sulphates, and even with known amounts of both cysteine and brombenzene injected the excretion may be by ethereal sulphates, bromphenylmercapturic acid or both. These crystals, then, do not represent a quantitative test, but only indicate the presence of mercaptan bodies.

Fig. 8.—Crystals of bromphenylmercapturic acid from the urine of a jaundiced animal.

In the hope of eliminating cysteine from jaundiced dogs with this drug, a third group of four dogs was selected; their common ducts were tied as before, and they were placed on a protein diet (fig. 9). Shortly after jaundice appeared they were given 0.1 cc. of brombenzene per kilogram each day by mouth. The same clinical picture developed as in the other dogs on protein feeding, but after from seven to ten days they were much more active and playful. Their postoperative course was protracted, being practically double that of dogs fed protein without brombenzene. They showed no signs of bleeding but died of emaciation, inanition and a moderate degree of hepatic cirrhosis. At autopsy a small amount of blood was found in the terminal bowel of

two animals. One animal in this group received, in addition, ultraviolet irradiation for an hour each day. He not only remained in better condition than the other animals throughout the experiment, but lived longer than any in the group. The urine was collected weekly, acidified, allowed to stand ten days, alkalinized, filtered, redissolved in acid, passed through animal charcoal, taken up with a weak solution of ammonia and evaporated in order to isolate crstals of bromphenylmercapturic acid.[44] These were found early in the stages of jaundice when brombenzene was given, but they did not appear late in the disease. We

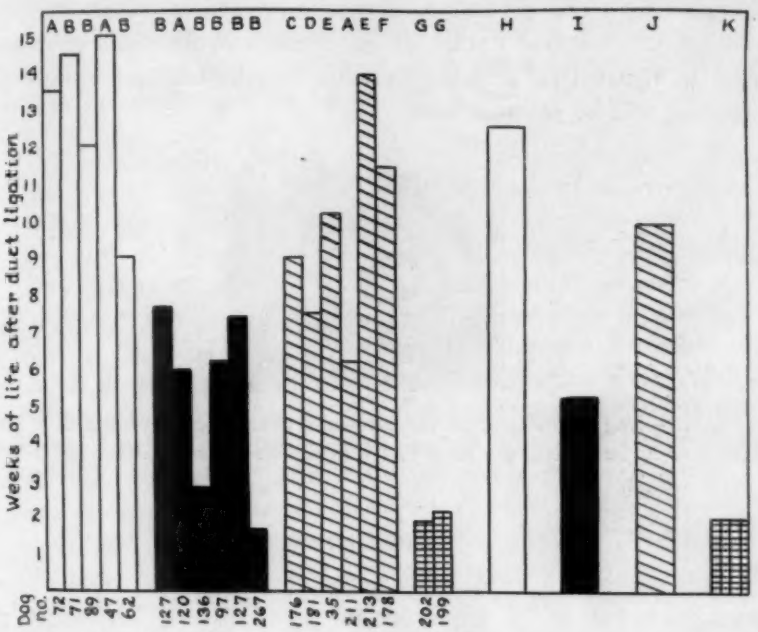

Fig. 9.—Longevity chart of jaundiced dogs. The white areas represent a balanced diet; the black, a meat diet; the oblique lines, a meat diet plus 0.1 cc. of brombenzene daily per kilogram of body weight; the squares, a meat diet plus 0.4 cc. of brombenzene daily per kilogram of body weight. The primary causes of death were as follows: *A*, cirrhosis; *B*, hemorrhage; *C*, undetermined; *D*, general edema and slight bleeding; *E*, volvulus; *F*, cirrhosis and slight bleeding; *G*, brombenzene poisoning. Charting *H* to *K* represents the average duration of life in jaundiced dogs: *H*, those on a mixed diet; *I*, those on a meat diet; *J*, those on a meat diet plus 1 cc. of brombenzene daily per kilogram of body weight; *K*, those on a meat diet plus 0.4 cc. of brombenzene daily per kilogram of body weight.

attributed this to the fact that the synthesis of bromphenylmercapturic acid, a function of the liver, was inhibited by blockage of bile, and that the intermediate ethereal sulphates only were synthesized.

The Grote test was positive in each animal but did not reach the intensity shown in the untreated dogs. The maximum intensity corresponded to 1 + or 2 +.

Figure 10 shows an animal at the end of three months after ligation of the duct. During this time he had been given a meat diet with 0.1 cc. of brombenzene per kilogram of body weight. He showed emaciation from extreme toxicity but no tendency to bleed.

While brombenzene in large doses produces the same toxic lesions as other benzene compounds, the doses used here have caused no apparent histopathologic changes or physiochemical reactions other than those mentioned. Complete studies of the changes in the tissue and in the blood in obstructive jaundice, cysteine intoxication and brombenzene poisoning will be reported later.

Fig. 10.—Dog at the end of three months after ligation of the duct during which time he had received a meat diet with 0.1 cc. of brombenzene per kilogram of body weight.

COMMENT

With the experience of other investigators and the results of our own work before use, it seems that the hemorrhage which follows prolonged obstructive jaundice is not due to quantitative deficiency in the measurable formed clotting elements of the blood, but rather to an addition of some inhibiting substance which changes the quality of the fibrinogen, fibrin and whole clot. This substance does not, as was popularly supposed, always prolong the coagulation time, for we have found consistently normal or short coagulation time in practically all of the hospitalized patients and experimental animals. Furthermore, the hemorrhage in these subjects is not a massive and sudden exsanguination, but rather a slow consistent oozing which over a period of hours results in serious loss of blood. This slow type of bleeding is most commonly observed in experimental animals in whom small amounts of blood start to ooze into the gastro-intestinal tract through

the mucosa for sometimes from eight to ten days before fatal hemorrhage occurs. This is also occasionally seen in hospitalized patients, but of course in them bleeding occurs more often following operations on the extrahepatic biliary system. The bleeding in these types, however, is similar and is a slow oozing type of exsanguination rather than an acute hemorrhage. The influence of operation in inducing this bleeding is apparently important, for patients and experimental animals that would not have bled otherwise bleed when channels of the circulatory system are artificially opened. The effects of damage to the tissues during operation and of anesthesia on this bleeding are apparently also contributory. Whether these effects are due to an introduction of added intermediate products of protein metabolism into the circulating plasma because of inhibited hepatic excretion, or to a reduction of the efficiency of hepatic metabolism, we are not prepared to say, but it is probable that both occur.

The mechanism of bleeding, as we have observed it, both in vitro with the addition of cysteine to normal blood and in vivo by injecting cysteine into normal animals and by inducing a collection of metabolites by ligation of the common duct, is one of seepage through an inefficient clot. As previously stated, the blood clots as rapidly or more rapidly than normal. The clot which is formed, however, is influenced by the collected intermediate products of protein metabolism, and, instead of forming a strong type of occluding mesh which retracts and efficiently prevents the escape of blood, a porous, nonretractile, friable gel of poor consistency is precipitated which is not capable of obstructing the flow of blood and through which both cells and plasma pass slowly. This is most evident after the initial retraction of very small vessels has relaxed. Cysteine and the related forms of mercaptan have this ability to influence the plasma and clotting elements, and that they do collect in excessive amounts in obstructive jaundice may be adequately demonstrated by colorimetric reactions. This theory is further strengthened by the behavior of subjects with such obstructions when brombenzene medication is used, and its effect in improving the clotting mechanism is interesting and definite. It is evident, however, that the animals fed suitable amounts of brombenzene are not protected to any extent from the toxic effects of cholemia but that their longer period of survival is due to the fact that they do not bleed.

Last, but quite important, is the fact that protein diets are apparently badly suited to subjects with biliary obstruction, and that high amounts of carbohydrates are beneficial. We have not yet had an opportunity to carry out this preoperative regimen on patients with obstructive jaundice who are about to be operated on, but we hope to report this procedure in connection with a number of operative cases at a later date.

CONCLUSIONS

1. There is no important quantitative change in the formed clotting elements in obstructive jaundice, but the quality of fibrinogen and fibrin is definitely altered.

2. The contention that sedimentation time is an index to clotting power is not substantiated.

3. The sedimentation time appears to vary inversely as the amount of fibrinogen.

4. The clotting time is normal or shortened, with few exceptions, and is not a reliable index to clotting power.

5. The clot formed is large, friable and nonretractile.

6. The cause of this clotting deficiency is the accumulation of incompletely metabolized products of proteins in the blood.

7. These products are sulphur-containing compounds, probably cysteine and its related mercaptans.

8. The use of brombenzene effectively reduces the amount of circulating cysteine and definitely reduces the bleeding tendency in experimental animals.

ATLANTO-EPISTROPHEAL SUBLUXATIONS

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NEW YORK

Subluxations of the cervical vertebrae were first described in this country in 1889 by Walton,[1] who devised the method of manipulative reduction that bears his name. In 1907 Corner [2] reported twenty cases of atlanto-epistropheal subluxations and dislocations, all of traumatic origin, and gave what is probably the best account of the mechanism of their production. The first report of a case of distention subluxation, i. e., subluxation probably due to effusion in the joints concerned, was made by Jacobs [3] in 1916. In 1932 Watson Jones [4] reviewed fourteen cases from the literature and reported two personal cases. The present paper is based on a consideration of twenty cases of distention subluxation and ten cases of related conditions observed from 1923 to 1933 at the New York Orthopaedic Dispensary and Hospital.

ANATOMIC CONSIDERATIONS

The atlas and epistropheus articulate through four joints: the atlantodental joint between the anterior atlantal arch and the dens epistrophei; the transversodental joint between the transverse ligament and the dens, and the two joints between the lateral masses.[5] Often the synovial cavities of all four joints communicate with each other and with the atlanto-occipital articulations. When this monocele type of synovial cavity is present inflammatory processes or effusions in one joint have free access to the other joints.

The joints drain into the retropharyngeal glands, into which the nasopharynx also drains. The efferent vessels of these glands drain into the deep cervical chain, as do the tonsil, the middle ear, the teeth and the nose. A pathway to the joints is thus suggested from foci about the head and neck. The second cervical nerve runs for a portion of its course in the capsule of the atlanto-epistropheal joint, which offers a probable explanation of occipital neuralgia in many cases in which this joint is affected.

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* Fellow of the New York Orthopaedic Dispensary and Hospital.

1. Walton, G. L.: Boston M. & S. J. **120**:277, 1889.
2. Corner, E. S.: Rotary Dislocations of the Atlas, Ann. Surg. **45**:7, 1907.
3. Jacobs, C. M.: Atlas and Axis Luxation, Am. J. Orth. Surg. **16**:357, 1916.
4. Jones, R. Watson: Spontaneous Hyperaemic Dislocation of the Atlas, Proc. Roy. Soc. Med. **25**:58, 1932.
5. Poirier, P., and Charpey, A.: Traité d'anatomie humaine, Paris, Masson & Cie, 1926, vol. 1, p. 74.

The opposed articulating surfaces of the lateral masses are both convex. When denuded of cartilage the inferior facets of the atlas are slightly concave. In vivo, however, this concavity becomes a convexity by virtue of the greater thickness of the cartilage in the middle (1.5 mm.) as compared with that in the periphery (0.5 mm.). A similar distribution of the cartilage increases the convexity of the superior articular surfaces of the epistropheus. Hence, when the articulation is examined from its lateral aspect it resembles the end-views of the hulls of two boats placed keel to keel. The anterior and posterior spaces, where the surfaces of the two joints do not come into contact, are occupied by a fringe of synovial membrane. When the atlas is fully rotated on the epistropheus, and only the peripheral part of the articular cartilages are in contact, the cervical portion of the spine loses from 2 to 3 mm. in height. This arrangement allows a shorter capsule to serve

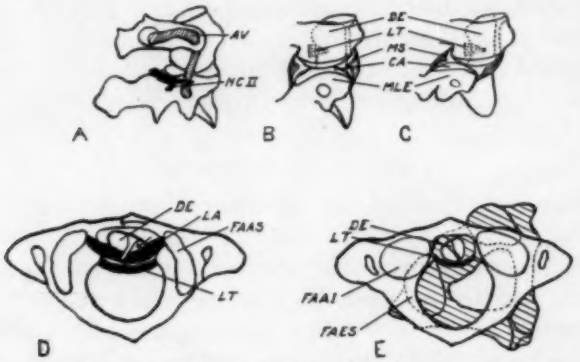

Fig. 1.—*A*, lateral view of the atlas and epistropheus; *B*, lateral view looking directly into the atlanto-epistropheal joint; *C*, same as *B*, with the epistropheus rotated; *D*, superior view of the atlas and dens; *E*, superior view of the atlas and epistropheus with rotation of the epistropheus. The contiguous surfaces of the joints are represented. The drawings are diagrammatic: *AV*, vertebral artery; *NCII*, second cervical nerve; *DE*, dens; *LT*, transverse ligament; *MS*, synovial membrane; *CA*, articular cartilage; *MLE*, lateral mass of the epistropheus; *LA*, alar ligament; *FAAS*, superior articular surface of the atlas; *FAAI*, inferior articular surface of the atlas; *FAES*, superior articular surface of the epistropheus.

the joint. The synovial fringes in the joint, when inflamed or adherent, may conceivably be an obstacle to the reduction of a subluxation or to free motion in an unluxated joint. Possibly these fringes are responsible for the nuchal crepitus elicited on rotation in most cases of rheumatoid arthritis.

The anterior atlantal arch is maintained in close apposition to the dens in all positions by a strong, thick, fibrous band, the transverse ligament, which passes between two tubercles prominently situated in the middle of the internal aspect of each atlantal lateral mass. The total

thickness of the cartilage of the atlantodental joint is from 1 to 1.3 mm. When the atlantodental interval is greater than this distance, there is a corresponding stretching or relaxation of the transverse ligament.

Movements of rotation at the atlanto-epistropheal joint are checked mainly by the alar ligaments, attached to the posterolateral portion of the head of the dens, running laterally and superiorly to the internal aspect of the occipital condyles. Figure 1 *D* illustrates how, as the atlas rotates to the left about the vertical axis of the dens, the right alar ligament becomes taut toward the limit of rotation and causes the head to be tilted to the right. This mechanism explains how rotation of the head is accomplished much more easily when accompanied by tilt in the opposite direction and why this tilt is present in rotation deformities.

The upper cervical vertebral foramina are relatively large to allow for the narrowing of the neural canal that occurs with rotation. In the average adult the dimensions of the vertebral foramen of the atlas are 30 mm. transversely and 25 mm. sagittally; of the epistropheus, 25 and

TABLE 1.—*Mobility of Different Portions of the Cervical Spine*

	Flexion and Extension (Total), Mm.	Lateral (to Each Side), Mm.	Rotary (to Each Side), Mm.
Atlanto-occipital	20 to 50	7 to 20	0
Average	30	10	
Atlanto-epistropheal	10	4	40
Remainder of cervical spine (5 joints)	30	30	40

19 mm.; and of the third cervical vertebra, 23 and 15 mm. The cord is 10 mm. in diameter in this region.

Table 1 showing the mobility of the different portions of the cervical spine is based on the textbook on anatomy by Poirier and Charpey,[5] confirmed by roentgenographic studies of my neck and clinical observations. The mobility is somewhat greater in children and is less in older subjects. Rotation is decreased in hyperextension and hyperflexion and is increased in slight flexion. Since there is no rotary mobility between the head and the atlas they rotate as a unit.

MECHANISM OF SUBLUXATION

A distinction must be made between subluxation of the atlas on the epistropheus and rotation deformity occurring in the atlanto-epistropheal joint. With a simple rotation deformity the transverse ligament is not relaxed, and there is no slipping forward of the atlas on the epistropheus. However, the rotation of the atlas on the epistropheus is fixed and necessitates a compensatory rotation in the lower part of the cervical spine to bring the head to the neutral rotary position. There are usually

tilt and flexion, along with rotation, deformities. Rotation deformity represents fixation in a position possible to a normal neck.

Subluxation implies relaxation or stretching of the transverse ligament and the slipping forward of the atlas on the epistropheus. Between these vertebrae is normally 10 degrees of flexion extension mobility, accomplished by a rocking motion of the convex surfaces of the joints on each other. The transverse ligament prevents any forward gliding of the atlas. With relaxation of the ligament, however, one or both of

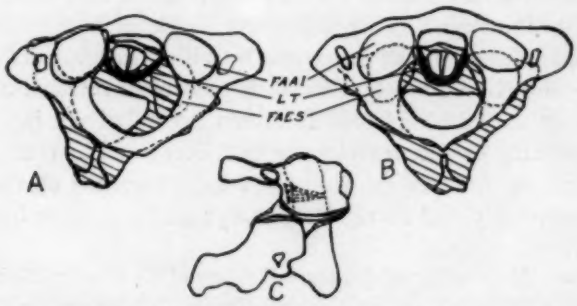

Fig. 2.—*A*, unilateral subluxation of the atlas; *B*, bilateral subluxation of the atlas; *C*, lateral view of the atlas and epistropheus showing subluxation of the atlas with flexion. The drawings are diagrammatic.

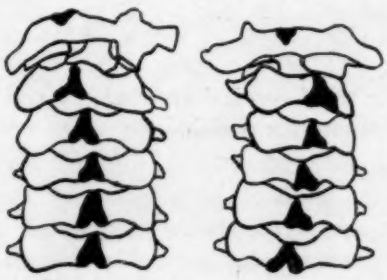

Fig. 3.—Diagrams of the cervical vertebrae viewed posteriorly in a normal rotation to the right and in right rotation deformity. In the latter the head and atlas are brought to the midline through a rotation of the cervical vertebrae to the left from the second segment downward.

the atlantal lateral masses may slip forward on the epistropheus. Flexion of the atlas on the epistropheus usually accompanies this subluxation. If the subluxation is unilateral or predominantly on one side rotation deformity is also present.

Complete dislocation of one or both atlantal lateral masses is impossible without dangerous narrowing of the neural canal, unless there is associated fracture of the dens. An examination of an articulated atlas and epistropheus reveals that in order for dislocation to occur when

both the dens and the transverse ligament are intact, 65 degrees of rotation is necessary. In the adult the neural canal is thereby narrowed to a diameter of 7 mm. If the transverse ligament is relaxed sufficiently to allow a forward subluxation of 5 mm., a complete dislocation of one lateral mass can take place with 45 degrees of rotation, the neural canal being thereby narrowed to a diameter of 12 mm. All the observed forward subluxations of more than 5 mm. were bilateral. With an intact dens a complete bilateral forward dislocation obliterates the neural canal altogether.

Posterior subluxation of the atlas as a whole is manifestly impossible without fracture of the dens or of the anterior arch of the atlas. If the transverse ligament is not relaxed any rotation forward on one side results in a corresponding rotation posteriorly on the other.

When the head becomes fixed in a position of rotation through the atlanto-epistropheal joint, with or without subluxation forward, the subject attempts to bring the head to a position of "eyes front" by rotating through the lower cervical joints in a direction opposite to the rotation deformity. Each of the vertebrae can rotate about 8 degrees to either side. The compensatory rotation occurs from the second cervical vertebra down, that is, the second presents the maximum rotary deviation, the lower vertebrae successively approaching the neutral position. The spines of the cervical vertebrae thus rotated deviate to the side of the midline toward which the head is turned, which is the reverse of what occurs when the head is turned on a normal neck (fig. 3).

A similar mechanism produces lordosis of the lower cervical spine to compensate for the flexion component of the deformity when that is marked.

ROENTGENOGRAPHIC OBSERVATIONS

The lateral and anteroposterior views referred to in this section signify roentgenograms centered on the atlas, using the following technic: a lateral view centered just below a line through the tips of the mastoid processes, and an anteroposterior view taken through the wide-open mouth centered along a line connecting the upper teeth and the occipital protuberance.

Lateral View.—1. Increased Anterior Atlantodental Interval: This sign is pathognomonic of forward subluxation of the atlas, and its extent and persistence are important in judging the progress of the condition and in making a prognosis. Forward displacement is gaged by the distance between the anterior border of the dens and the ring shadow which marks the anterior arch of the atlas. Normally this interval should not be more than from 1 to 2 mm.

2. Swelling of Soft Tissue: The depth of the shadow of the soft tissue anterior to the first two cervical vertebrae should normally be

equal to that of the shadow of the soft tissue anterior to the other cervical bodies. Thickening of this shadow in subluxated or deformed cervical vertebrae indicates active inflammation and suggests the need of functional rest for the neck.

3. Flexion of the First on the Second Cervical Vertebra: The line joining the inferior borders of the anterior and posterior arches of the atlas and the line joining the inferior borders of the articular processes of the epistropheus with the inferior border of its spinous process should be parallel or converge posteriorly when the head is in the neutral position. With the head flexed these lines may normally

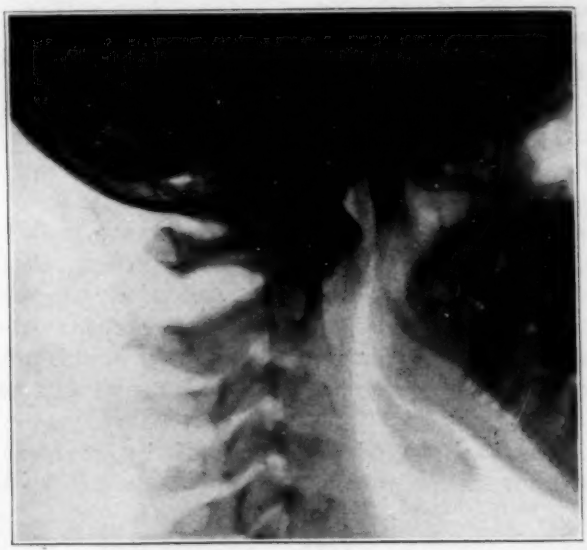

Fig. 4.—Lateral view of the atlantal region in a case of subluxation. Flexion of the atlas is not present. The anteroposterior view taken at the same time showed no overlapping. Note the increased anterior atlantodental interval, the shadow of the soft tissue anterior to the vertebral bodies, the rotation of the second and third cervical vertebrae and the slight lordosis.

converge anteriorly at an angle as great as 10 degrees. In cases of subluxation and rotary deformity of the first on the second cervical vertebra the atlas is usually somewhat flexed on the epistropheus. In only two cases in this study was hyperextension present. If a true lateral view of the atlas is not obtained it is difficult to determine the degree of flexion.

4. Compensatory Lordosis of the Lower Cervical Vertebrae: This sign is present when the flexion of the first on the second cervical vertebra is marked, usually in cases of severe bilateral forward subluxation.

5. Failure of the Two Halves of the Posterior Arch of the Atlas to be Superimposed: In a true lateral view the two sides of the posterior arch are superimposed. Since there is lateral tilt deformity in cases of unilateral forward subluxation, and often in simple rotation deformity, lateral roentgenograms taken at right angles to the neck rather than to the atlas will show both sides of the posterior arch.

6. Evidence of Rotation of the Cervical Vertebrae from the Second Vertebra Downward: This is seen in the presence of rotation deformity between the atlas and epistropheus. The joint spaces of the lateral articulations of the two sides are no longer superimposed, but their shadows are projected, one anterior to the other.

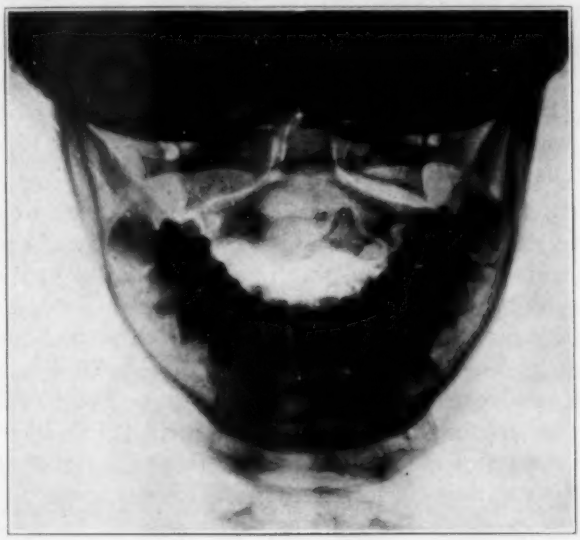

Fig. 5.—Anteroposterior roentgenogram of the atlantal region. Note: the deviation of the chin and the spine of the epistropheus to the same side of the midline; overlapping on the side of the forward movement of the atlas; the apparent lateral displacement of the atlas. That the displacement is only apparent is evidenced by the symmetry of the atlantal lateral masses about the dens.

7. Diminished Anteroposterior Diameter of the Atlantal Vertebral Foramen: George and Leonard [6] in their work on roentgenography of the spine stress the importance of diminished width of the atlantal vertebral foramen in a diagnosis of forward subluxation of the atlas. This sign is open to much misinterpretation, since rotation of any part of the cervical spine gives rise to foreshortening, which simulates diminished width of the foramen.

6. George, A. W., and Leonard, R. D.: The Vertebrae Roentgenologically Considered, volume 8 of Annals of Roentgenology: A Series of Monographic Atlases, edited by James T. Case, New York, Paul B. Hoeber, Inc., 1929, p. 81.

Anteroposterior View.—1. Lateral Deviation of the Epistropheal Spine: This is the most important sign to be looked for in the anteroposterior view. The shadow of the spine has the form of an inverted V and is readily identified. When normal cervical vertebrae in a position of rotation are roentgenographed in the sagittal plane of the head, the shadows of the spines of the vertebrae are successively farther from the midline. If, however, there is rotation deformity of the head, with or without forward subluxation, the spine of the second cervical vertebra is found on the same side of the midline as the direction of rotation, while the spines of the lower cervical vertebrae are successively nearer the midline. The reason for this deviation to the side toward which the head is turned has been previously referred to: The lower cervical vertebrae, in an attempt to enable the patient to look straight ahead, rotate in a direction opposite to that of the rotation deformity. The amount of rotation present between the atlas and epistropheus can be estimated. If the spine of the second cervical vertebra is in line with the lateral margin of the dens the amount of rotation is 15 degrees; if with the transverse process of the atlas, 50 degrees.

2. Overlapping of the Shadow of the Lateral Masses of the First and Second Cervical Vertebrae on One or Both Sides: As indicated in the mechanism of subluxation, this sign is rarely due to the complete downward and forward dislocation of one lateral mass of the atlas on the epistropheus. Bilateral overlapping is often seen, but a bilateral dislocation with an intact dens is incompatible with life. The two factors that produce the appearance of overlapping are flexion of the atlas on the epistropheus and a forward or backward position of one or both lateral masses of the atlas, due either to rotation or to subluxation forward. Both of these factors were present in every case in this series showing overlapping in the anteroposterior view. An appearance of overlapping can be obtained if a roentgenogram is taken with the head flexed and rotated. This was true of my neck, and an appearance of overlapping of the lateral masses on one side was obtained. Furthermore, three cases in this series without forward displacement of the atlas showed overlapping. On the other hand, three cases showing forward displacement of the atlas did not present overlapping. In several other cases the overlapping originally present was absent in subsequent roentgenograms while the forward displacement remained.

In two cases in this series the anteroposterior roentgenogram represented the lateral mass of the atlas which was rotated posteriorly as overlapping the corresponding lateral mass of the epistropheus; e. g., in two roentgenograms of one patient taken on the same morning, with no change in the deformity, overlapping was present on one side only in each roentgenogram, but on opposite sides. This fact suggests that the side on which the overlapping appears depends on the variation in the

direction of the projection of the rays through the spaces of the joints. When the roentgen rays are projected from a point inferior to that employed in the usual technic it is on the posteriorly rotated side that overlapping is seen.

To sum up, overlapping is an appearance occurring with flexion and rotation of the first on the second cervical vertebra. It is not pathognomonic of subluxation or of the side on which the forward movement has taken place.

3. Lateral Displacement of the Atlas: This sign is subject to many errors of interpretation. The first cervical vertebra has a normal lateral mobility on the second as great as that seen in roentgenograms of most subluxations. Moreover, whenever rotation is present the second cervical vertebra is foreshortened in the anteroposterior view, giving the appearance of lateral displacement to one or both sides. In order to give significance to this appearance of lateral displacement the head must be held erect on the spine and both lateral masses must be displaced laterally with respect to the dens. Under these conditions a unilateral displacement represents a pathologic fixation in a position within the normal range of motion. A bilateral displacement of the atlantal lateral masses in opposite directions, however, indicates the fracture of one of the arches of the atlas.

Decalcification of the upper cervical vertebrae, emphasized by Jones,[4] was not found in any case in this series. The only pathognomonic sign of subluxation in the absence of a fracture is an increased anterior atlantodental interval in the lateral view of the atlas.

ETIOLOGY

An etiologic classification of the subluxations and rotation deformities studied divides them into three groups: (1) secondary to distention of the joints with relaxation of the transverse ligament, twenty cases; (2) traumatic, nine; (3) resulting from poliomyelitis, one.

Distention Subluxations.—The age distribution in this group was: between 5 and 10 years, eight; between 10 and 15 years, seven; between 15 and 20 years, two; 20 years and over, three.

As a rule the patients were pale, undernourished and sickly. Eleven of the twenty patients had rheumatic fever; two of these died of it while under observation. Fifteen patients, eight of whom had rheumatism, had inflammatory processes about the head and neck shortly before the onset of symptoms of subluxation. The inflammatory conditions which occurred were tonsillitis, mastoiditis, cervical abscess, osteomyelitis of the lower cervical vertebrae, acute dental infection, chronic coryza and influenza. Eight of these patients had operations shortly before the onset of symptoms. It has been thought that twisting of the head while

a patient is under anesthesia might produce a subluxation. However, in half the cases the onset of symptoms was a day or more after the operation and was gradual and progressive. Furthermore, no subluxations were observed following any other type of operation than those for an inflammatory process in the head or neck. The operations performed were mastoidectomy, tonsillectomy, incision and drainage of a cervical abscess and removal of tuberculous glands of the neck (followed by suppuration).

The following hypothesis is advanced to explain the occurrence of distention subluxations: The synovial membrane of the atlanto-epistropheal joints responds either to a generalized attack of rheumatic fever or to infection carried through the lymphatics or by continuity from infectious processes about the head or neck. This response to infection occurs most readily in persons who have had rheumatic fever. The joint between the dens and the transverse ligament is simultaneously affected, possibly because it often communicates with the other joints of the articulation. The transverse ligament relaxes and permits forward displacement of the atlas on one or both sides.

Traumatic Conditions.—Rotation deformities without subluxation were occasioned most frequently by trivial traumas, such as falling out of bed or turning the head suddenly. Overcorrection of congenital torticollis, with immobilization in plaster following myotomy, was responsible for two cases. The condition is probably analogous to sprain. In no case in this series did trauma alone precede forward subluxation. In two cases fracture of one of the atlantal arches with lateral subluxation of the lateral masses resulted from forces applied suddenly to the top of the head. One patient was buried in a cave-in; the other struck the bottom of a swimming pool when diving.

Poliomyelitic Subluxation.—Paralysis of the stabilizing muscles of one side of the neck gave rise in one case to true subluxation of the atlas with relaxation of the transverse ligament.

SYMPTOMS

Subluxation and simple rotation deformities of the atlanto-occipital joint present a similar symptomatology. The chief complaint was most often of stiffness, frequently of deformity or pain on motion. These three symptoms were always present to some degree. Rarely, and only in the severer cases, were difficulty in opening the mouth, dysphagia and changes in the voice complained of. Three patients with bilateral subluxation had neurologic symptoms due to pressure on the cord. Occipital headache was recorded three times.

Difficulty in opening the mouth is associated with, and is probably due to, extreme flexion of the head on the neck in marked subluxations

Swallowing becomes difficult apparently because of the obstruction offered to the rising of the glottis by the flexion of the head and the protrusion of the second, third and fourth cervical vertebrae into the pharynx as they hyperextend to compensate for the flexion deformity in the atlanto-epistropheal joint.

SIGNS

The head is held stiffly in flexion with the chin close to the neck. When rotation deformity is present in addition to flexion the head is held tilted toward, and rotated away from, the side on which the atlas has moved forward. The patient appears to be straining to bring the head to the midline. On arising from the prone position, the head is supported by the hands. Instead of turning the head, the whole body is turned.

The spine of the second cervical vertebra is palpated on the side toward which the head is turned, i. e., opposite the side on which the atlas has moved forward. The mechanism of this phenomenon has been previously referred to. The paraspinal muscles stand out prominently on the side toward which the epistropheal spine is deviated and are usually in spasm. A colleague described a case in which this spastic muscular mass was mistaken for an abscess. The patient died during attempted drainage. If the subluxation is symmetrically bilateral the spine of the epistropheus is not rotated, but becomes more prominent in the midline. The soft tissues in the region of the subluxation are tender, particularly over the bony prominences.

Mobility is extremely limited by spasm. Attempts at motion cause pain, usually localized just below the mastoid processes. In the supine position, with the head supported on the table, the mobility is often much greater, probably because the stretched transverse ligament is no longer under tension. Even when lying down it is usually impossible to rotate the head past the midline toward the side on which the atlas has moved forward.

When rotation deformity is present, examination of the pharynx reveals a bulging on the side opposite that on which the atlas has moved forward. This is caused by the rotation of the cervical vertebrae below the first (previously referred to).

The neural canal is narrowed in proportion to the amount of forward subluxation of the atlas. In five patients the subluxation was over 10 mm. Three patients with subluxation of 11, 12 and 14 mm., respectively, had signs of pressure on the cord. On the other hand, two patients with subluxations of 12 and 13 mm., respectively, did not have pressure on the cord.

The heart should always be examined in view of the high incidence of rheumatic fever in this group.

DIAGNOSIS

An acquired torticollis with spasm of the muscles should at once arouse suspicion of subluxation or rotation deformity at the atlanto-epistropheal joint. A history of rheumatic fever, infection about the head or neck or trauma antecedent to the onset of the symptoms is still more suggestive. Final diagnosis should be made by roentgenogram.

Differential diagnosis should distinguish the condition from congenital torticollis, specific or nonspecific arthritis of the cervical spine (a subluxation may develop later on an arthritic basis), myositis and fibrositis of the neck, spasmodic torticollis, osteomyelitis of the cervical vertebrae and subluxations of the lower cervical vertebrae (particularly following trauma).

TREATMENT

The aim of treatment in these conditions is twofold: (1) reposition of the bones concerned in their normal relationship and (2) restoration of the inflamed or damaged structures of the joint to a healthy state.

From the therapeutic point of view the cases studied can be divided into four groups: distention subluxation; simple rotation deformity; fracture of the atlas, and poliomyelitic subluxation.

Distention Subluxation.—Of the twenty cases in the group reduction was attempted in fourteen. The methods employed were traction and manipulation, of the Walton type, i. e., tilt, then rotation opposite to the deformity, with fixation in a plaster cast for from four to twenty weeks. Traction consisted of: (1) straight traction on the head in bed; (2) strong traction on the head with pelvic countertraction, followed by support in a plaster cast; (3) a plaster cast applied while the head is under traction, and (4) suspension with manipulation.

No permanent reduction of the forward displacement was obtained under treatment except in one instance. Strong traction on the head with pelvic countertraction followed by the wearing of plaster bandages for twenty weeks effected a reduction of the forward slipping from 12 to 3 mm. On the other hand, follow-up examinations revealed that in nine patients, two of whom had been treated by massage only, reduction of the subluxations occurred spontaneously (incomplete in six and complete in three).

Recumbency alone relieves the spasm considerably, but is more effective when combined with traction. However, straight traction in bed has proved totally ineffective in diminishing the forward displacement of the atlas unless the head is hyperextended.

Roentgenograms taken while heavy traction was being exerted on the head showed separation of the cervical vertebrae, but when the traction acts in the direction of flexion it has no influence in lessening the forward subluxation.

Jacobs,[3] who first reported cases of distention subluxation, noted while checking his manipulation by digital examination of the pharynx of one patient that the prominence of the subluxated lateral mass of the atlas disappeared when the head was hyperextended and promptly reappeared when the head was flexed. Berkheiser and Seidler [7] used traction with hyperextension in their five cases of distention subluxation and secured reduction in all of them.

Hyperextension is an important principle in the treatment of this condition. Not only do the movements of the vertebrae when hyperextended represent the reverse of the movement by which the subluxation took place, but recurrence of the deformity is prevented by the tension produced on the anterior spinal ligament, which then acts as a splint to the vertebrae. Light traction in hyperextension secured reduction within a week's time in two patients now under treatment and not included in this series.

Reduction accomplished, the head and neck should be rested for a sufficient time to allow for a subsidence of the inflammation and restoration of the transverse ligament to its normal length and strength. Subsidence of the inflammation can be judged clinically from the disappearance of muscle spasm, and roentgenographically from the disappearance of swelling of the soft tissue. The speed of restoration of the transverse ligament depends on the age of the patient, the length of time the subluxation has been present, the extent of the subluxation and the patient's general condition. Rest is most readily secured by immobilization in plaster in hyperextension. The period of immobilization might be varied, depending on the conditions mentioned, from three weeks to five months. Immobilization should never be maintained in flexion, and the position should be checked roentgenographically. A short period in bed to allow the muscles to regain their tone should follow removal of the support. A check-up by roentgen examination should be made shortly after the patient is allowed to be out of bed without support.

Simple Rotation Deformity.—In the eight cases in this group reduction with restoration of function was accomplished regardless of the form of treatment applied. Treatment employed included: massage only (two cases), wearing of a Thomas collar for two weeks, traction in bed for eighteen days, wearing of a plaster helmet for five weeks (two cases) and Walton manipulation with the helmet for five weeks.

One patient whose deformity resulted from overcorrection of a congenital torticollis following a myotomy was completely and permanently relieved by one day's traction in hyperextension.

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7. Berkheiser, E. J., and Seidler, F.: Nontraumatic Dislocations of the Atlanto-Axial Joint, J. A. M. A. **96**:517 (Feb. 14) 1931.

The same principles of treatment probably apply to this group as apply to distention subluxations, but, owing to the minor nature of the injuries sustained by the joint structures, immobilization following reduction is brief or may be omitted altogether.

Fracture of the Atlas.—Anatomic reduction of a fractured atlas is impossible by any method known at present. Relief from spasm of the muscles and the trauma of bearing weight by traction in bed is indicated for such a period as will allow union to take place, and protection from motion by a suitable collar after the bearing of weight is deemed safe.

PROGNOSIS

The length of follow-up in the series varied from three months to seven years, with an average of two years. Patients were regarded as well if they had no symptoms, no clinical deformity and a good range

TABLE 2.—*Results in Eighteen Cases of Distention Subluxation*

	Well	Improved	Unimproved
A. Age 5 to 10	5	3	0
10 to 15	3	1	1
15 to 20	0	2	1
Over 20	0	0	2
B. Original forward displacement			
5 mm. or under	6	3	0
Over 5 mm.	2	3	4
C. Reduced	3	0	0
Partly reduced	5	2	0
Unreduced	0	4	4

of motion in all directions without spasm; improved if there was any definite lessening of the original symptoms or any definite improvement in mobility; unimproved if there was negligible or no improvement in symptoms and physical signs.

The subluxations were classified as reduced, partially reduced and unreduced. The anterior atlantodental interval was measured in millimeters, and the subluxation was considered reduced if this distance was 2 mm. or less. All reductions with the exception of one partial reduction were spontaneous and were not secured under treatment.

Table 2 gives a summary of the results of treatment in eighteen cases of distention subluxation.

It may be concluded that the younger the patient is the smaller the amount of the original forward displacement, and the greater the amount of reduction secured the better are the results. In only one case was there any increase in the subluxation six months after it was recognized. It is assumed that after the acute inflammation has subsided there is little tendency to further stretching of the transverse ligament.

All the patients with simple rotation deformity made a complete recovery.

The two patients with fractured atlantal arch recovered a good range of motion, but both continued to suffer from pain in the neck, particularly in damp weather and on resuming activity after resting.

SUMMARY

Nonfatal dislocations of the atlas without fracture of the dens must be rare. Subluxation of the atlas forward is not uncommon and is associated with relaxation of the transverse ligament. Subluxation is often accompanied by rotation deformity, but rotation deformity often exists without subluxation. Roentgenographically, subluxation is diagnosed by the increased anterior atlantodental interval; rotation deformity, by the deviation of the spine of the epistropheus. Effusion in the atlanto-epistropheal joints, resulting from rheumatic fever or from foci of infection nearby, causes the relaxation of the transverse ligament and the subluxation. Simple rotation deformity is usually caused by minor trauma. Pain, stiffness, deformity, limitation of motion in the neck and spasm are found in both subluxations and rotation deformities. Bilateral subluxation is characterized by flexion of the head and a prominent epistropheal spine in the midline. Unilateral subluxation and rotation deformity are characterized by flexion, rotation, tilt and deviation of the epistropheal spine to the side toward which the head is turned. Traction in hyperextension will reduce both subluxations and rotation deformities. Immobilization should last long enough to allow recovery of the damaged structures of the joints, especially the transverse ligament. Subluxations show little tendency to increase six months after the original slipping; rather they tend to decrease spontaneously. The younger the patient and the less marked the subluxation, the better the prognosis.

CONCLUSION

Patients with an acquired torticollis, especially when giving a history of rheumatic fever or infection about the head and neck, should be studied roentgenographically. If an atlanto-epistropheal subluxation or rotation deformity is found, traction with hyperextension followed by immobilization in hyperextension is the treatment of choice.

LOCAL ATROPHY OF BONE

III. EFFECTS OF VITAMIN D AND OF CALCIUM ON LOCAL ATROPHY AND UNION

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Convalescence after fractures of and operations on bone is prolonged because bone heals more slowly than does soft tissue and because the immobilization required during the process of healing leads to local atrophy of the bone and muscles and to a decrease in the range of motion permitted by the soft tissues of the part. If the union of bone could be hastened and if local atrophy could be prevented considerable economic advantage would result and a proportionately larger number of patients could be treated in orthopedic hospitals each year with the same number of beds. In this article we report a series of clinical experiments in which therapeutic doses of vitamin D and of calcium were administered to normal children receiving an adequate diet in an attempt to hasten union and to prevent local atrophy of bone after stabilizing operations on the feet.

We know of no observations on the effects of added vitamins and calcium on local atrophy of bone in animals receiving an adequate diet, but a considerable literature has accumulated on the effect of these substances on the union of bone. There is general agreement that if animals are given a scorbutic diet the union of fractured bones will be delayed or will fail to occur, depending on the severity of the avitaminosis.[1] However, an excess of vitamin C administered to an animal receiving a normal diet has no effect on the healing of fractures (Israel and Frankel,[2] Schilowzew,[3] Roegholt [4] and Holasz and Marx [5]).

From the Department of Surgery of the Washington University School of Medicine and the Shriners' Hospital for Crippled Children, St. Louis.

1. Watanabe, T.: Experimental Researches on Effect of Avitaminosis on the Healing of Bone Fractures, Virchows Arch. f. path. Anat. **251**:281, 1924.

2. Israel, A., and Frankel, R.: The Influence of Vitaminosis on the Healing of Fractures, Klin. Wchnschr. **5**:94, 1926.

3. Schilowzew, S. P.: The Influence of Avitaminosis, Deutsche Ztschr. f. Chir. **209**:320, 1928.

4. Roegholt, M. M.: The Influence of A, B, C and D Vitamin and the Elements of Calcium and Phosphorus on the Healing of Fractures, Arch. f. klin. Chir. **168**:783, 1931.

5. Holasz, G. V., and Marx, Josef: Is Callus Building Influenced Through the Addition of Vitamin C? Arch. f. klin. Chir. **169**:121, 1931.

Observers who have added vitamin D and calcium to an adequate diet have reported conflicting results. Bors [6] and Collazo [7] found that the addition of vitamin D to the diet increased the size of the callus and the rapidity of the union of fractures in rats as compared with control rats which were given an adequate diet. Roegholt [4] found that an insufficient amount of vitamin D caused delayed union, but made no observations on the effect of an excess of vitamin D in the diet. Moritsch and Krammer [8] and Pincussen and Newmann [9] administered a substance which contains vitamins A, B, C and D, calcium and phosphorus to experimental animals and to patients with fractures, and concluded that this substance caused fractures to unite more rapidly than did similar fractures in animals and patients receiving a normal diet. On the other hand, Hellner,[10] Vara-Lopez,[11] Swart [12] and Lewis [13] found that the addition of an excess of vitamin D to an adequate diet had no effect on the healing of fractures, and Tammann [14] found that the addition of a large excess of vitamin D to the diet of rabbits with experimental fractures had a deleterious effect on the callus and in some instances caused atrophy or fracture of the callus.

After we had found that atrophy of bone could be demonstrated in roentgenograms of a foot which had been immobilized in a plaster of paris cast and had gained a fair idea as to the amount of atrophy which might be expected to occur after eight weeks of immobilization, we decided to use postoperative roentgenograms of the feet as criteria in determining whether or not the atrophy could be prevented by adding vitamins and calcium to the diet.

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6. Bors, E.: Vigantol and Fracture Healing, Zentralbl. f. Chir. **54**:3266, 1927.

7. Collazo, J. A.; Rubina, P., and Varela, B.: The Rôle of Vitamin D in the Regeneration of Bone Tissue and Callus Building in Experimental Fractures, Arch. f. klin. Chir. **158**:214, 1930.

8. Moritsch, P., and Krammer, E.: The Influence of a Calcium and Phosphorus Containing Vitamin Preparation (Vito Phos) on Callus Building, Wien. med. Wchnschr. **79**:1454, 1929.

9. Pincussen, and Newmann: The Relation of Fracture Healing to Chemical Changes in the Blood and the Influence of Vitamins and Calcium on These Changes, Arch. f. klin. Chir. **165**:483, 1931.

10. Hellner, Hans: The Influence of Vigantol on Fracture Callus in Healthy Animals, Deutsche Ztschr. f. Chir. **209**:307, 1928.

11. Vara-Lopez, R.: Experimental Researches on the Influence of Fracture Healing, Deutsche Ztschr. f. Chir. **212**:101, 1928.

12. Swart, H. A.: The Effect of Irradiated Ergosterol on the Healing of Experimentally Produced Fractures in Animals, J. Bone & Joint Surg. **12**:360, 1930.

13. Lewis, K. M.: Non-Effect of Irradiated Ergosterol in the Treatment of Fractures, Ann. Surg. **92**:415, 1930.

14. Tammann, H.: Retardation of Fracture Healing by Vitasterin, Arch. f. klin. Chir. **165**:473, 1930.

For the purpose of our studies we selected children in good general health who were to be subjected to stabilization operations on one or both feet. In most instances the operation was the classic Hoke operation consisting of a subastragaloid arthrodesis and a plastic osteotomy of the neck of the astragalus with an arthrodesis of the astragaloscaphoid joint. In some instances the calcaneocuboid joint was subjected to arthrodesis (triple arthrodesis). Thus we had a series of patients with a fairly uniform surgical procedure in whom we could

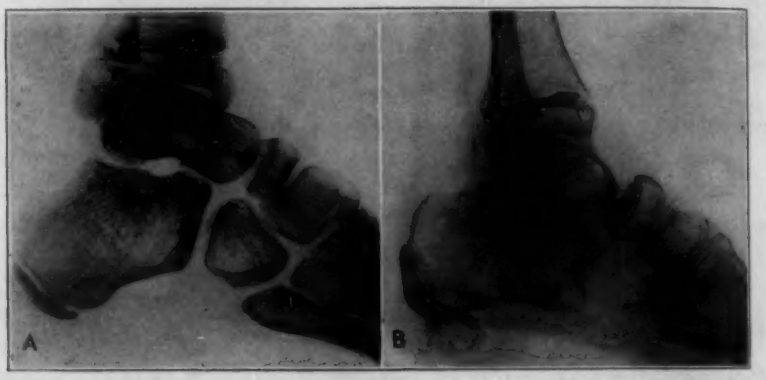

Fig. 1 (B. Z.).—The left foot: *A*, before the operation, and *B*, eight weeks after a Hoke operation and immobilization in a plaster of paris cast. This patient received cod liver oil twice a day during the period of immobilization.

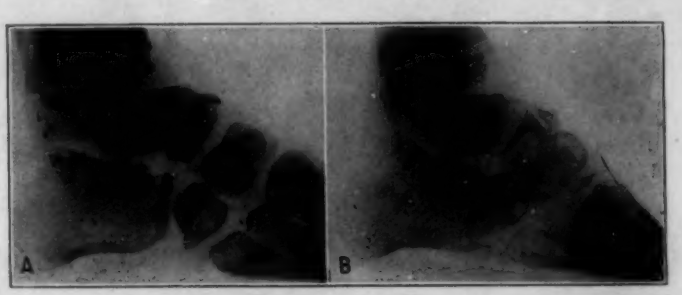

Fig. 2 (A. W.).—The left foot: *A*, before immobilization, and *B*, after operation and immobilization for ten weeks in a plaster of paris cast. This patient received cod liver oil and calcium during the period of immobilization.

study the degree of atrophy and the rapidity of union between the bones at the healing joints. After the operation it is customary to keep the foot and leg immobilized in a plaster cast which extends from the middle of the thigh to the toes. The immobilization is continued for eight weeks. Then the cast is removed and the patient begins to walk in an ordinary shoe.

Forty patients were studied. Ten of these were used as controls and received the ordinary hospital diet at the Shriners' Hospital for

Crippled Children, which is an adequate diet for normal children. The other thirty patients received the same diet, but in addition, ten received viosterol (20 drops daily), seven received cod liver oil [15] (2 teaspoonfuls daily) and the remaining thirteen received cod liver oil and calcium (2 teaspoonfuls of cod liver oil and 2 teaspoonfuls of calcium compounds—about 1 Gm. of calcium phosphate daily).

At periods of from seven to eight weeks after the operations the casts were removed, lateral roentgenograms were made of the feet, and the feet were examined clinically. Other than some separation of the edges of the skin, which is a frequent occurrence after these operations, there were no complications in the series, and complete healing had taken place at the end of the usual eight weeks' period. No difference in the healing was noted when the controls were compared with the patients who received the added vitamin D and calcium. The same was true of the stability of the feet. On clinical examination and functional tests the feet of the controls appeared to be as stable and as firmly united as the feet of those who had received the added vitamin D and calcium.

When the roentgenograms of the feet were studied and compared with preoperative roentgenograms of the same feet, and when those of the controls were compared with those of the patients who had received the added vitamin D and calcium, it was found that there was about the same degree of atrophy of bone and of roentgenologic evidence of union of bone in both series.

COMMENT

The observations recorded in this article indicate that local atrophy of bone is the result of local conditions and that it cannot be prevented or appreciably lessened by an abundance of vitamin D and calcium in the diet. Likewise, the healing and union of bones appear to require a certain amount of time, and we found no evidence that the addition of vitamin D and calcium to an adequate diet will reduce this time.

It is to be emphasized that all our patients received an adequate diet and our results are not to be interpreted as evidence that patients convalescing from fractures and operations on the bones should not receive vitamin D and calcium. It is probable that a diet deficient in vitamin D and calcium would lead to increased local atrophy and delayed union of bone.

It is further to be noted that therapeutic doses of vitamin D and calcium do not cause increased local atrophy or delayed union of bones.

CONCLUSION

The addition of vitamin D and calcium to an adequate diet does not lessen local atrophy of disuse or accelerate the rate of union of bones.

15. The cod liver oil contained 20,000 vitamin A and 10,000 vitamin D units to the ounce (29.5 cm.).

A REVIEW OF UROLOGIC SURGERY

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(*Concluded from page 170*)

PROSTATE GLAND

Carcinoma.—Young [27] stated that carcinoma of the prostate gland generally begins as a nodule just beneath the posterior capsule, where it is easily palpable by way of the rectum. Hypertrophy of the prostate gland almost never begins in this region, and the two may develop concurrently, each occupying a different part of the gland; in fact, 50 per cent of patients with carcinoma of the prostate gland also have adenomatous hypertrophy, not infrequently quite remote from and entirely uninvolved by the carcinoma. Later the carcinoma spreads in all directions, ultimately penetrating the fibrotic capsule of the adenomatous enlargements and finally completely replacing the nonmalignant cells within such capsules.

Young described his radical operation for the care of the prostate gland: After exposing the gland through an inverted V incision by the usual division of the central tendon and recto-urethral muscle, it is opened through the membranous urethra, a retractor is introduced and the posterior surface of the gland is exposed. The radical operation is begun by transverse division of the urethra, isolation of the prostate gland from beneath the anterior transverse fascia, exposure of the bladder near the prostate gland and resection of the cuff of the bladder

27. Young, H. H.: The Ultimate Results in the Treatment of Carcinoma of the Prostate by the Radical Removal of the Prostate, Vesical Neck and Seminal Vesicles, J. Urol. **29**:531 (May) 1933.

with transverse division of the trigon about 1 cm. below the urethral orifice. The bladder is then pushed up, care being taken not to injure the ureters, and the ampullae and vesicles are isolated, clamped, divided and ligated high up. Careful hemostasis must be obtained. The new high frequency coagulation machine is of great assistance, but the deep pedicle of the seminal vesicles should be ligated and all serious bleeding stopped. No difficulty is experienced in anastomosing the bladder to the membranous urethra, a portion of the anterior wall being used and the remainder closed longitudinally. A retained urethral catheter takes care of drainage. The deep angles of the wound are lightly packed with a long strip of iodoform gauze, which is brought out through one corner of the wound, the rest of the wound being closed by interrupted sutures of waxed silk.

Young stated that this operation, which at first seemed so formidable, has been carried out in 42 cases at this clinic; it has also been carried out in 40 cases by G. G. Smith at the Massachusetts General Hospital, who stated that one and a half hours suffice for the operation, that the mortality rate is no greater than in prostatectomy for benign hypertrophy, and that healing is accompanied by less infection and is often quicker. Young's statistics indicate that of 4,295 operative prostatic specimens, 460, or 10.7 per cent, revealed carcinoma. If cases were included in which treatment was with radium, the percentage of carcinoma would be higher. These statistics demonstrate the great importance of recognizing carcinoma and recognizing it early.

The average age of Young's 42 patients, for whom the radical operation for carcinoma of the prostate gland was employed, was 64 years; the youngest patient was 51 years of age and the oldest, 77. The predominant symptoms were: frequency, nocturnal emission, complete retention, urethral discharge, burning on urination, hesitancy, hematuria, urgency and difficulty in completely emptying the bladder; several patients made no complaint. The anesthetic agent employed was ether in 9 cases, and oxygen and ether in 15. Anesthesia was caudal in 15 cases, and spinal in 3. The average number of days in the hospital was forty-two. The mortality rate was 4 deaths, or 9.5 per cent; in the last 37 cases the rate was 2 deaths, or 5.4 per cent. Twenty-seven patients have been traced who left the hospital after the radical operation, now more than five years ago. Of these, 11 of the original 42 patients, or 26 per cent, have lived five years without recurrence. Eighteen patients are living, only 3 with recurrence, at periods varying from one to seventeen years after operation. Ten of these patients reported perfect urinary control.

Young stated that the principal object of his explanation of the results was to demonstrate, first of all, that early diagnosis of carcinoma of the prostate gland is generally easy. A very hard nodule, palpable

by rectum, unless shown to be a stone by a roentgenogram, should be looked on with grave suspicion. In such a case the patient should be subjected to perineal operation at which the lesion can be seen, palpated and, if necessary, incised and studied microscopically. If physicians could be taught to make rectal examinations much more frequently and to be suspicious of every markedly indurated prostate gland, even when only a small nodule is present, many patients would be brought to early radical operation and to ultimate cure. Young stated that freedom from recurrence can be obtained in a large percentage of cases, and that excellent functional results with complete nocturnal control can be secured in practically every case; he also stated that diurnal control can be obtained in a large percentage of the cases when the patient is on his feet.

Smith [28] stated that in early prostatic carcinoma, before the growth has extended beyond the capsule of the gland, the possibility of cure is encouraging, but there are few cases in which the condition is discovered while patients are in this stage. When the growth has developed beyond this point, the question of cure is no longer pertinent; the object of treatment then is to relieve urinary obstruction, if that exists, and to retard, as much as possible, further growth. Smith reviewed 25 cases of carcinoma of the prostate gland in which high voltage roentgen treatment was given. Eighteen of the patients suffered from pain in the back, legs or pelvis; all of them were relieved following irradiation. This relief varied in degree from partial to complete, and in duration from one to six months. One series of treatments frequently relieved pain for three months. The prompt relief from pain was sometimes remarkable. The effect of irradiation on the prostate gland itself was less pronounced. In 9 cases the gland appeared to be smaller after treatment; in 10 it continued to increase in size, and in 2 it gave no evidence of change. The degree of urinary obstruction was not affected.

Smith reviewed the results of treatment with radium in 29 cases of carcinoma of the prostate gland. In 3 cases treatment consisted in insertion of seeds or needles through the perineum; in 11 cases partial perineal prostatectomy was performed with implantation of radium in the remaining shell of prostatic tissue, and in 16 cases radium was implanted through the open bladder. In most of these cases the obstructing median bar was removed at the same time; in 2 others, radium was implanted in the tips of vesicles after total perineal prostatectomy. Smith reported that in 29 operations, there were 6 operative deaths, or 21 per cent. Three were due to embolus, 2 to cardiac failure and

28. Smith, G. G.: The Treatment of Cancer of the Prostate, New England J. Med. **208**:57 (Jan. 12) 1933.

1 to sepsis. Sixteen patients died of carcinoma, the average length of life after operation being thirteen months. Of 6 patients known to be alive, 1 has lived four years. He is incontinent and has sclerosis of the vesical neck and a bladder that holds only 1 ounce (30 cc.). He is losing weight, and although roentgenograms do not show bony metastasis, he looks cachectic. Three other patients are having considerable trouble, and 3 patients, who were operated on recently, although they have good control, have strangury and pain at the neck of the bladder.

Smith felt that of all surgical methods of attacking carcinoma of the prostate gland, total perineal prostatectomy is the only really aggressive one. He also reviewed his surgical cases for evidence of malignant disease of the prostate gland; he has operated on 42 patients in the last thirteen years. In all cases, the diagnosis has been established by microscopic examination. There were 3 operative deaths. Seventeen patients died of carcinoma, with an average duration of life of twenty-seven months. Four patients are alive but give evidence of local recurrence or metastasis in the spinal column. None of these has any urinary obstruction. Seventeen patients are alive and apparently well. Of 13 who are alive and free from recurrence or metastasis, 6 have lived five or more years since operation; of the 11 who died without recurrence or metastasis, 5 lived five or more years. Smith concluded by saying it is his conviction that in early cases of prostatic carcinoma total prostatectomy should be done.

Smith, in a second article,[29] stated that perineal prostatectomy for carcinoma is not a particularly difficult operation to perform. He has found that the time required to perform this operation is about an hour and a half. It can generally be done with one application of spinal anesthesia, particularly if nupercaine is used. He stated that convalescence has been unusually easy; in fact, it is less fraught with anxiety than after ordinary prostatectomy because there seldom is any bleeding, and there seems to be no toxic absorption, or at best very little, from the space from which the prostate gland is removed. Since 1918, Smith has performed 41 of these operations. Of the patients operated on, 4 died in the hospital, and 13 who survived the operation died of carcinoma of the prostate gland. The average length of time between operation and death was twenty-one months; the longest, seven and a half years. In 2 cases, in both of which death finally resulted from carcinoma originating in the prostate gland, the patients, as far as they knew, had been well between five and six years after operation; local recurrence then developed with metastasis, and the end came fairly rapidly.

29. Smith, G. G., in discussion on Young,[27] p. 571.

An important point is that in these cases there are no local symptoms in regard to difficulty of urination after operation.

Smith reported that several of the papillary tumors which he encountered were of the type in which the neck of the bladder is involved and the tumor is malignant, as had been proved by biopsy and cystoscopic inspection. With improvement of the modalities of high frequency current, Smith found that by using a flexible-loop electrode he could ream out the whole neck of the bladder and the tumor without any difficulty, whereas in all these cases operation previously had been by the suprapubic route. In another type of case, which has presented difficulties in the past and necessitated laborious, frequent treatment for large papillomas, Smith, with the same type of electrode, has been able to cut down the tumors (tumors of enormous size) so that within two or three treatments the whole tumor has been destroyed, the superabundant mass being cut away with the loop electrode and the base immediately coagulated with the ordinary button electrode.

Transurethral Resection.—Bumpus [30] stated that in the sixteen months from Jan. 1, 1932, to July 1, 1933, at the Mayo Clinic, 488 patients were subjected to transurethral resection of the obstructing portion of the prostate gland. Only 1 patient of this group died. In a recent study, 600 cases in which prostatectomy was performed were compared with 600 cases in which resection was done; 43 per cent of the patients who were subjected to prostatectomy had been prepared by permanent drainage with a catheter, whereas only 24 per cent of the other group were so prepared. Of these 1,200 cases, suprapubic cystostomy was performed in 45 per cent of the cases in which the patients were to undergo prostatectomy and in 19 per cent in which the patients were to undergo transurethral resection.

Bumpus reported that when transurethral resection for prostatic obstruction was first undertaken, only those cases were included in which obstruction was minimal. The field has gradually enlarged, however, until, in the first six months of 1933, only 3 patients underwent suprapubic enucleation of the gland, whereas 202 patients underwent transurethral resection. Bumpus felt, therefore, that, with few exceptions, prostatic obstruction can be corrected by transurethral methods if the urethra permits the passage of instruments. He stated that transurethral resection has been criticized as a more or less temporary measure. He reported that from Jan. 1, 1925, to Jan. 1, 1932, he had removed tissue which, on microscopic examination, revealed adenofibromatous hypertrophy in 96 cases. In 1 case in which operation had been performed in 1925, 8 Gm. of tissue was removed, a considerable amount for those

30. Bumpus, H. C., Jr.: Preparation for, and Care Following, Transurethral Resection of the Prostate Gland, Urol. & Cutan. Rev. **37**:674 (Oct.) 1933.

days. When last traced, 86 of these patients were alive and 68 were satisfied with the results of operation, many after a lapse of more than four years. From January 1927 to July 1933, subsequent prostatectomy was required in only 9 (1.3 per cent) of 702 cases in which transurethral resection had been performed. During the same period, transurethral resection has been employed in 21 cases in which prostatectomy had been performed previously.

Walker [31] discussed the anatomy and physiology of the vesical neck and pointed out that resection is rendered more difficult by the fact that the knowledge of its anatomy and physiology is so incomplete. An examination of the muscle fibers of the vesical neck would indicate, in Walker's opinion, that opening of the neck of the bladder is brought about not merely by relaxation of the sphincter but also by the dilating action of certain longitudinal fibers passing through this structure. In discussing pathologic changes in the vesical neck, Walker made use of the term "dysectasia," previously introduced by Legueu, to indicate the condition in which there exists some obstacle to the opening of the neck. He then discussed the histology of obstruction of the vesical neck under the following divisions: (1) glandular hyperplasia, (2) muscular hypertrophy, (3) increase of fibrous tissue, (4) increase of more than one tissue and (5) malignant disease. After stating that the aim of operation is to remove all tissue that interferes with normal opening of the neck, Walker considered the various routes by which the neck may be approached, namely, the perineal, the suprapubic and the transurethral. The operation of cuneiform excision of the posterior lip of the internal meatus was then described as well as the more radical operation of complete excision. He then described transurethral methods, starting with the history of transurethral operations and ending with a description of instruments in present use. He pointed out that modern transurethral methods are of three kinds: simple fulguration, the coagulated tissue being left to slough away subsequently; electrocoagulation followed by immediate punching out of coagulated tissue; removal of obstructing tissue by means of the McCarthy loop. Walker said that in his experience sepsis is a grave danger in all cases in which coagulated tissue is left behind, and for that reason he expressed a strong preference for operations that include immediate removal of coagulated tissues. In his opinion, this removal may best be accomplished by the use of the McCarthy loop which removes tissue at the same time as it seals off bleeding points.

Walker stated that since the aim of surgical intervention is to remove enough tissue to restore the ability of the neck to dilate, transurethral

31. Walker, Kenneth: The Surgery of the Bladder Neck, Fifth Cong. Internat. Soc. Urol., 1933, p. 516.

operation must be employed only when, in the opinion of the operator, this aim can satisfactorily be achieved by such methods. When surrounding structures are extensively involved, as, for example, in a case of fibrous "bar" associated with a fibrous prostate gland, the more radical operation of excision of the neck will probably be required.

Scholl [32] stated that transurethral prostatic resection is unquestionably a procedure that will be used extensively in the future. The recognition of early obstruction and treatment by resection while the patient is still in good health will greatly reduce the incidence of advanced prostatism and its need for extreme surgical procedure. He stated that prostatic resection is definitely not a minor operation. It carries a high potential risk, even in the hands of an expert, and it should be done only after thorough examination and adequate preparation by a urologist specially skilled in the technic. The ready acceptance of this procedure by physicians for their own personal needs suggested to him a successful future for the method.

Weijtlandt [33] stated that disturbances in evacuation of the bladder due to lesions of its neck may be classed under the clinical name "prostatism." In the great majority of cases such disturbances are caused by adenoma of the submucous periurethral glands (prostatic hypertrophy) or by carcinoma of these glands or of the prostate gland (prostatic carcinoma) or by sclerotic processes fundamentally of an inflammatory nature. The cause of urinary retention in such cases is not, as a rule, the purely mechanical "cut-off" of the urinary passage; a catheter, for instance, can usually be passed. The most common cause of retention is some obstacle to the functioning of the sphincter in the process of opening. This is due partly to mechanical fixation of the muscle by pathologic tissue and partly to secondary hypertony of the sphincter.

Weijtlandt reported that the various operative measures applied, such as prostatectomy, extirpation of the vesical neck, cuneiform excision, punch operation, electrocoagulation and "cutting current" resections, are all the same on one point, namely, removal of a portion of the pathologic tissue from the region of the vesical neck and sphincter. The success of this procedure depends on whether sufficient tissue is removed to allow what remains of the vesical neck and of the prostatic urethra to regain a sufficient degree of flexibility, or, at all events, to prevent the sphincter from being impeded in its function of opening the vesical neck. There are three methods of transurethral treatment which are well known: Young's punch operation, or one of the modified forms of the Braasch-

32. Scholl, A. J.: An Evaluation of Transurethral Prostatic Resection, West. J. Surg. **41**:278 (May) 1933.

33. Weijtlandt, J. A.: On the Surgery of the Bladder Neck, Fifth Cong. Internat. Soc. Urol., 1933, p. 477.

Bumpus, McCarthy or Day and Phélip operations; Caulk's cautery-punch operation, and the "cutting-current" resection methods of Stern-Davis, McCarthy, Canny Ryall and of von Lichtenberg. Weijtlandt concluded that the results thus far reported do not warrant any definite conclusions. Publication of more detailed reports on more cases will be required to make it possible to form a solid judgment regarding these therapeutic methods, the indications for and the practical performance of them, the attendant danger and their efficacy over long periods of time.

Electrosurgical Resection.—Belt, Charnock, Folkenberg and Falconer [34] described a current activator for prostatic resection which is amply powered, compact and inexpensive. It has an undamped current which is supplied by vacuum tubes and which is suitable for cutting under water. A spark-gap apparatus furnishes a damped current for the coagulation of bleeding vessels.

A series of experiments were carried out which indicated that the optimal density of the radio frequency current for adequate under-water cutting was 300 volts and 0.7 amperes. It was discovered that the death of cells occurred for an appreciable distance beyond the actual cut if the density of the current was increased to 325 volts and 1.8 amperes. Healing subsequent to cuts made with such a current proceeded along the cut edge without the death of an appreciable amount of tissue beyond the point of contact with the advancing loop. These investigators further discovered that a coagulating current of low wattage, when held in contact with the surface of the prostatic urethra for a length of time sufficient to whiten the tissues, caused death of cells to great depths. The depth of the subsequent slough bears a direct relation to the length of time the active electrode is in contact with the tissues.

Disease of the Vesical Neck.—Marion [35] stated that from a symptomatic point of view it is evident that there are two types of disease of the vesical neck. In the first, or so-called congenital type, the manifestations appear in early childhood; in the second type, the trouble arises at a later age, without ever having been preceded by other disturbances. In the congenital form, complications occur early in infancy. The child is obliged to use pressure when urinating. He takes a long time to urinate, and this is sometimes noticed by the parents. The symptoms then increase more or less rapidly. They are comparable in every way with the manifestations of prostatic hypertrophy and vary slightly according to the patient; there is, sometimes, incomplete retention with-

34. Belt, Elmer; Charnock, D. A.; Folkenberg, A. W., and Falconer, R. A.: The Current Activator in Electro-Surgical Resection of the Prostate, Urol. & Cutan. Rev. **37**:687 (Oct.) 1933.

35. Marion, G.: Maladie du col vésical, Fifth Cong. Internat. Soc. Urol., 1933, p. 451.

out distention, distinguished by more or less pronounced pollakiuria and the presence of residual urine in more or less abundant quantities. This incomplete retention occasionally leads to distention with incontinence. Finally, and frequently, the dysuric stage is abruptly intensified by complete retention.

The acquired type of disease of the vesical neck has symptoms identical with those of the congenital type, which in turn are identical with those of prostatic hypertrophy; in these cases, however, it has been impossible to trace dysuric trouble in infancy. The symptoms appear at a more or less advanced age and develop rapidly. In such cases there invariably are urethral or periurethral antecedents, such as long-standing chronic urethritis, often accompanied by periurethral suppuration and stricture.

BLADDER

Tumors.—Sindoni,[36] in a case of primary adenocarcinoma of the bladder, studied the vesical mucosa with reference to the existence of vesical glands, a subject about which there is no unanimity of opinion today. Albarran gave the most detailed description of these glands, which he divided into two groups: The first group of glands are near the trigon and consist of small depressions, which do not have a membrana propria, only a simple wall of connective tissue lined with an epithelium not essentially different from that of the epithelial cells of the bladder. These glands do not secrete mucus but contain cellular detritus due to superficial epithelial desquamation. The second group is situated outside of the neck of the bladder, especially on the anterior wall, and the glands are analogous to those of the first group, and still rudimentary. Contrary to Albarran's opinion and that of investigators who agreed with him, there are a number of other investigators who think these formations are not glands but only glandlike depressions.

In view of such differences of opinion, Sindoni took up the histologic study of these structures for himself in healthy bladders, examining only the trigon. He found that whereas the gross appearance of these structures was somewhat varied, the histologic details were uniform: at times there were superficial excavations of the epithelium, and in other cases simple or ramified tubular formations, but the epithelium in all was exactly the same as that of the surface and the connective tissue had the same structure. In fact, the tunica propria followed the outlines of the glandlike formations and was composed of fibrillar connective tissue and elastic fibers. Sindoni concluded that both in children and in adults there are solid epithelial masses and simple or branching tubular formations at the vesical trigon which in their structure must be regarded

36. Sindoni, M.: Glandule vesicali e tumori a struttura glandulare della vescica, Arch. ital. di urol. **10**:309 (July) 1933.

as minute extroflexions of the mucosa. Despite the fact that the gross appearance of these structures is that of glands, he thinks such a name cannot be given to them, because the term "gland" is closely bound up with the idea of secretion, and there was no histologically observable secretion. A mucicarmine test gave negative results. Sindoni denied the existence in the bladder of any true mucous glands, and he thought that to explain the genesis of local glandular neoplasms "of cylindric epithelium" the view must be taken that these are in some cases tumors starting in the prostate gland and in others heterotopic epithelial inclusions, or else that they are a process of metaplasia of the vesical epithelium.

[COMPILER'S NOTE.—The subcervical group of glands of the vesical neck and region of the trigon have been recognized as a clinicopathologic entity since Albarran's time and, as is evident from the literature, their existence as true glandular formations has been confirmed by many investigators. Lowsley, in a study of a series of postmortem examinations, discovered that they were present in 32 per cent of the cases. Likewise, these subcervical glands or tubules have been recognized as an entity, since they tend to cause adenomatous hypertrophy, infection and other pathologic conditions which produce obstruction at the outlet of the vesical neck. The subcervical glands of Albarran may likewise undergo adenocarcinomatous hypertrophy independent of the prostate gland itself, and they may thus give rise to a pedunculated tumor of the bladder. Folsom described them in relation to the urethra and vesical neck of the female and, more recently, Gutierrez, in a review of the literature, explained how they may produce pathologic conditions affecting the vesical neck.]

Carcinoma.—Watson [37] reported 411 cases of carcinoma of the lower portion of the urinary tract in which treatment was given at the New York Institute. In this study there were 204 cases of carcinoma of the bladder, 194 cases of carcinoma of the prostate gland, 11 cases of carcinoma of the urethra and 2 cases of carcinoma of a fistulous tract leading from the bladder. The cases of carcinoma of the bladder were divided into: (1) Carcinoma of the bladder, papillary or undifferentiated in type. In this group there were 32 cases; 5 patients were lost from observation after having been treated on an average of seven months after coming to the hospital, and 3 were not treated. Twenty-four patients were treated, and of these 3 were alive and gave no evidence of tumor a year and six months, two years and four months and ten years and one month, respectively, after starting treatment. Of the 21 patients who died, the duration of life after starting treatment had been an average of eight months. (2) Adenocarcinoma, an uncommon type of

37. Watson, E. M.: A Study of Carcinoma of the Lower Urinary Tract, J. Urol. **29**:545 (May) 1933.

vesical tumor arising from abberrant prostatic tubules, from the subtrigonal group of glands or from mucous glands occasionally contained within the wall of the bladder. This type of tumor was encountered twice; in both cases the patients were men, aged 50 and 54 years, respectively. (3) Epithelioma of the mucous membrane. Histologically, these tumors, although deeply infiltrating and rather rapidly growing, retain to a pronounced degree the characteristics of cells of the mucous membrane lining the bladder. In this group there were 134 cases. In 4 of these treatment was not given for various reasons. Of the remaining 130 patients, 90 are now dead, and of those who are living, 25 have no vesical tumor and 14 still have varying amounts. One patient was lost from observation at the end of a year and three months, but at the time gave no evidence of tumor. (4) Malignant papilloma, designated as such because of the papillary arrangement of cells. These tumors possess, however, all of the malignant properties of the previously mentioned types, but to a less degree. There were 24 patients with tumors of this type. Three of these patients were lost track of after having been under observation for from eight months to seven years with no tumor present at the last examination. Fourteen have died, 12 of malignant tumor. Seven of this group are still living; 6 of these have no tumor, and 1 has tumorous tissue present. Of the patients who died, the duration of life, on an average, was two years and one month after starting treatment. (5) Massive papillomatous tumors, the cells of which arouse suspicion but lack the earmarks of malignancy possessed by tumors of the other groups. There were 13 cases in this group, and in 4 of these the patients had been lost from observation with no evidence of tumor when they last were seen. One patient has died after one year and five months, and 8 are alive without evidence of tumor from one to seven years after treatment.

Watson stated that 194 patients with carcinoma of the prostate gland were treated; 65 of these had undergone previous operations for the removal of the gland, 4 by the perineal route and 61 by the suprapubic route, at a time varying from one to six years before entering the hospital. Of the 194 patients, 111, or 57 per cent, had carcinomatous infiltration of both seminal vesicles in addition to prostatic growth when they were admitted, whereas 30, or 15 per cent, had only one seminal vesicle infiltrated in addition to the prostatic growth. Twenty are now living, on an average of eleven months after beginning treatment; 1 of these has lived ten years and four months, and another died after eight years and one month. In this group no proved cure has been obtained.

Smith and Mintz [38] studied and reported 150 cases of tumor in the bladder. In this group, 117 operations had been performed, excluding

38. Smith, G. G., and Mintz, E. R.: Bladder Tumor, Am. J. Surg. **20**:54 (April) 1933.

simple cystostomies; 35 patients, or 30 per cent, died following operation. Smith and Mintz concluded that the grade of malignancy of the tumor of the bladder has no demonstrable relation to its tendency to metastasize. Squamous cell carcinoma metastasized almost twice as frequently as papillary carcinoma. They stated that one important cause of the poor results from surgical treatment of carcinoma of the bladder is the long delay between the occurrence of the first symptoms and the patient's entrance to the hospital.

Smith and Mintz stated that a comparison of the results of resection, electrocoagulation and radium implantation gives evidence that the lowest number of deaths in the hospital is among patients treated by electrocoagulation (26 per cent); resection revealed the next lowest number, 32 per cent, and radium implantation revealed the highest, 41 per cent. The end-results from these three methods were approximately the same. Smith and Mintz expressed the belief that no one method is suitable for all types of carcinoma. The surgeon should be prepared to employ whichever method seems best in the individual case.

Diverticulum.—Kutzmann [39] stated that diverticulum of the urinary bladder is a disease that affects chiefly the male, and nearly always it is associated with such an obstructive condition as prostatism or urethral stricture. The incidence of diverticulum is as follows: in urologic cases in general, 1.2 per cent; in cases of benign hypertrophy, 9.1 per cent; in cases of contracture and median bar obstruction, 16.8 per cent, and in cases of urethral stricture necessitating operation, 14.3 per cent. No definite clinical symptoms are noted, except, possibly, that if there is obstruction of the lower portion of the urinary tract with infection, an accentuation of the characteristic symptoms of the associated condition is present. Kutzmann stated that diverticulum of the urinary bladder is the result both of congenital and of acquired anatomic factors. He also stated that this condition can be treated most satisfactorily by correcting the obstructing factors, diverticula being treated individually only when they are of the retention type or of large size. This method of procedure will give gratifying relief from symptoms which have been intense and of long duration.

Cystostomy.—Goldstein [40] stated that emergency or early suprapubic cystostomy should always be performed when the catheter is giving unsatisfactory urinary drainage, when the urethra or bladder is ruptured, when the condition of the patient seems to be aggravated by the

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39. Kutzmann, A. A.: Diverticulum of the Urinary Bladder: Analysis of 100 Cases, Surg., Gynec. & Obst. **56**:898 (May) 1933.

40. Goldstein, A. E.: Emergency Suprapubic Cystostomy, J. Urol. **29**:609 (May) 1933.

presence of a urethral catheter and when patients with urinary obstruction have sensitive urethras.

Beer [41] enumerated the underlying causes of persistent and recurring suprapubic fistula following cystostomy. In the local group, the outstanding causes are improper surgical technic and partial prolapse of the vesical wall between the rectus muscles, gauze sponges left in the bladder, contracted bladder, overlooked large, infected diverticula and disharmony between the sphincter and the detrusor muscles. In the peripheral group, the causes of fistula are unrelieved obstruction at the neck of the bladder, fibrous vesical neck, adenoma and stones or stricture in the urethra. In the third group, central, Beer stated that infected kidneys, infected hydronephrosis and tuberculous kidneys are the main causes.

Injury.—Stevens [42] summarized the histories of 8 women whose bladders had accidentally been torn or incised during the course of a pelvic operation. In 2 cases, injury to the bladder had occurred during hysterectomy; in 2 cases during hysterectomy and salpingectomy, or oophorectomy or hysterectomy and both salpingectomy and oophorectomy; in 2 cases during salpingectomy and oophorectomy; in 1 case during tightening of the vesical sphincter, colporrhaphy, proctopexy and perineorrhaphy, and in 1 case during operation for excision of a sinus following cesarean section and freeing of adhesions to the uterus. The vesical wall was incised in 5 cases and torn in 3. Injury was detected at the time of operation in 7 cases and was not detected in 1 case; in these 7 cases, the wound was sutured, in 1 with drainage of the wound and in 1 with drainage of the bladder through a retention catheter. Convalescence was uneventful in 4 cases and prolonged in 3. Death resulted eventually from the vesical injury in 1 case as a result of bilateral pyonephrosis due to scar tissue obstructing the ureters following several attempts at repair of a vesicovaginal fistula.

Stevens concluded that infection of the urinary tract should receive appropriate treatment prior to operative procedures. The bladder and its attachments should be handled carefully during pelvic operations. Although no serious consequences follow repair of the vesical wall in the majority of cases, when injury is detected at the time of operation, exceptions to this rule are not uncommon. In cases in which drainage is used, convalescence is considerably shorter. Accidental operative injuries of the bladder are not infrequently responsible for prolonged convalescence, and they occasionally are fatal.

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41. Beer, Edwin: Postoperative Suprapubic Fistula: Analysis of Causes, Surg., Gynec. & Obst. **56**:959 (May) 1933.

42. Stevens, W. E.: Female Bladder Injuries Incident to Surgery, California & West. Med. **39**:388 (Dec.) 1933.

Injury Following Irradiation.—Dean [43] reported the cases of 47 women who were examined and treated for injuries of the bladder after irradiation of the uterus. Both radium and the roentgen rays were used, although by far the more intense radiation was delivered by radium. The primary uterine diseases comprised a number of conditions, both benign and malignant. Radiologic treatment varied within wide limits; it preceded the onset of urinary symptoms in the average case by two and a half years. Urinary symptoms consisted of frequency, dysuria and hematuria. Cystoscopic examination gave evidence of ulceration, anemic regions surrounded by intense inflammation or punctate hemorrhage. The lesion was situated in the posterior third of the vesical base in or near the median line. Diagnosis was based on the history and the results of vaginal examination, cystoscopy and biopsy. Treatment consisted of opiates for pain, lavage of the bladder with dilute phosphoric acid both transurethrally and by mouth, and instillations of mild silver protein and mercurochrome.

Dean concluded that when one employs the accepted methods of treating carcinoma of the uterus with high voltage roentgen rays combined with radium applied within the cervix or body, the vesical base, in some cases, unavoidably receives sufficient irradiation to cause ulceration. This possibility must be recognized by all physicians who treat uterine carcinoma. Vesical injury occasionally follows the application of comparatively light doses of irradiation to relatively insignificant uterine diseases. A woman who has reached the age when carcinoma is liable to develop, who suffers with frequency, dysuria and hematuria, and who presents on cystoscopic examination an ulcerated condition of the bladder, may have either vesical carcinoma or a tertiary reaction to irradiation. If an injury of the bladder due to irradiation is treated by irradiation, electrocoagulation, cauterization or by any of the other methods of treating carcinoma which are of a destructive nature, the results are disastrous. With proper management, however, the end-results are excellent in the majority of cases.

Nerve Control.—Mertz [44] stated that changes in the spinal cord resulting in disturbed nerve control of the bladder may be due to spina bifida occulta. Laminectomy, with freeing of the cord from pressure and traction, may relieve vesical symptoms. Laminectomy is justifiable in the presence of spina befida occulta when chronic urinary retention with distention of ureters and pelves coexists and no obstructive pathologic change is demonstrable. Laminectomy should not be carried out for enuresis alone, although definite spina bifida occulta exists, before

43. Dean, A. L., Jr.: Injury of the Urinary Bladder Following Irradiation of the Uterus, J. Urol. **29**:559 (May) 1933.

44. Mertz, H. O.: The Relation of Spina Bifida Occulta to Neuro-Muscular Dysfunction of the Urinary Tract, J. Urol. **29**:521 (May) 1933.

the patient has reached the age of puberty and then only after all other measures have failed. Mertz stated that of 6 patients who underwent laminectomy for spina bifida occulta, 2 were relieved of vesical symptoms and 2 gave evidence of slight improvement, whereas 2 obtained no appreciable relief from symptoms.

URETHRA

Tumors.—Counseller and Paterson [45] reviewed the literature and reported 12 cases of carcinoma of the female urethra. Four of the 12 patients were living five years after treatment, and those who died had an average duration of life of twenty-five and a half months; excluding 1 patient who lived six years, the average duration of life was sixteen months. The highest percentage of five year cures was obtained in cases in which radium and roentgen rays were used. Counseller and Paterson stated that obviously their series was too small to warrant conclusions with regard to results.

Lazarus [46] reported 3 cases of primary benign tumor of the urethra; 2 of the growths were papillomas and 1 was a fibromyoma. Among tumors of the urethra the polyps and papillomas represent epithelial tumors; the fibromyomas represent connective tissue tumors. Lazarus stated that polypi and cysts of the urethra are products of urethral inflammation rather than true neoplasms. Papillomas, although actual tumors, are frequently initiated by chronic inflammatory conditions within the urethra. Fibromas and fibromyomas are true neoplasms, the origin of which is not dependent on inflammation. The diagnosis of these tumors is usually made by inspection, either directly or through the cysto-urethroscope. Biopsy is frequently necessary for confirmation of the clinical diagnosis made by the eye. Lazarus said that the treatment consists of fulguration in cases of polypi, cysts or papillomas, or extirpation in cases of tumors of the connective tissue.

EPIDIDYMIS, SEMINAL VESICLES AND TESTES

Torsion of Spermatic Cord.—O'Conor [47] stated that most patients with acute torsion of the spermatic cord are seen first by their regular physicians who, as a rule, do not consider this condition in making a differential diagnosis. The diagnosis of orchitis, epididymitis or "acute hydrocele" is frequently made without proper correlation of history,

45. Counseller, V. S., and Paterson, Susanne J.: Carcinoma of the Female Urethra, J. Urol. **29**:587 (May) 1933.

46. Lazarus, J. A.: Primary Benign Tumors of the Urethra: Report of Three Cases, Urol. & Cutan. Rev. **37**:604 (Sept.) 1933.

47. O'Conor, V. J.: Torsion of the Spermatic Cord, Surg., Gynec. & Obst. **57**:242 (Aug.) 1933.

symptoms and physical signs. Since operative intervention results in conservation of the testis only when resorted to in the first few hours after the onset of symptoms, it is necessary that the diagnosis be made at the earliest possible moment.

Torsion may occur at any age; it has been recognized shortly after birth, and O'Conor stated that in one of his cases it occurred when the patient was 68 years of age. Probably it occurs more frequently during childhood and adolescence. Torsion of the spermatic cord has been observed more commonly on the right side and, in slightly more than half of the cases, in incompletely descended testes. It probably is brought about by contraction of the cremasteric fibers; there must be abnormal attachment of the testis and a certain deficiency in make-up of the gubernaculum associated with a more or less capacious tunica vaginalis. This anatomic variation would not cause torsion; the twist results from repeated contraction of cremasteric muscle bundles, strands of which may be anomalous. Therefore, the degree of torsion, that is, the number of half turns or full turns which the cord undergoes, depends indirectly on the freedom of the testis to be rotated inside the tunica and directly on the strength of the muscular contraction.

O'Conor stated that at operation the pathologic changes are characteristic. The cord above the twisted segment contains either dilated or flattened and partially obliterated spermatic veins depending on the extent of their occlusion in the twist. The spermatic artery is always greatly dilated but, usually, pervious. If the condition is acute, the surrounding tissue is edematous and, if chronic, it is adherent and fibrous. In acute cases, the twist occurs from without inward among more than two thirds of the patients. In the recurrent cases, no definite rule can be formulated. The extent of the twist varies from one-half turn to two full turns, and the site of the twist is always in that free portion of the cord which, covered by tunica vaginalis, suspends the epididymis and testis. O'Conor cited 9 cases, 5 of which were verified at operation, 3 by the observations and subsequent course and 1 by the obviously recurrent type of the condition with gradually developing atrophy. Orchitis, as a clinical entity, except in association with epidemic parotitis, is an uncommon condition, and the term should not be loosely applied to explain undifferentiated scrotal swellings. Bearing in mind the characteristic sudden onset of torsion, with the more or less typical local signs, will permit an early diagnosis to be made with immediate operative detorsion, which is necessary in most acute cases if testicular tissue is to be conserved.

Spermatocystitis.—Valverde [48] stated that in refractory cases of chronic spermatocystitis the only satisfactory treatment is the heroic

48. Valverde, Belmiro: Le lavage des vésicules dans le traitement des spermatocystites chroniques, J. d'urol. **36**:262 (Sept.) 1933.

one of lavage of the vesicles, either by means of Belfield's vasotomy or by the vasopuncture of Luys. Valverde prefers the latter, as it is simpler and gives somewhat better results. Lavage is done under local anesthesia with procaine hydrochloride or nupercaine, each vesicle receiving an injection of from 10 to 12 cc. of a 5 per cent solution of colloidal silver. In 8 of the cases there was a complication of simple funiculitis, which healed spontaneously. Valverde reported good results. The method can always be used successfully except when intrapelvic obstructions exist; under such conditions, the liquid cannot reach the vesicles and lavage must be done through the ejaculatory ducts by urethroscopic catheterization. Records of Valverde's service revealed 340 patients with chronic spermatocystitis among the 1,200 patients of this private clinic, and 452 patients with this condition among 2,800 patients with genito-urinary disease at the General Polyclinic of Rio de Janeiro. The technic is simple, though it demands great care: The isolation of the vas must be complete and careful to the last degree, pains being taken to liberate it from all sheaths and adhesions. When, however, there is obstruction, the procedure is more difficult; the vas must then be isolated over a distance of from 10 to 12 cm. in a search for a nonobstructive point. Puncture should be made in the middle of the canal, the hand of the operator following its direction rigorously. The injection must be made immediately, as soon as the resistance of the wall is overcome by the entrance of the needle. Absolute rest in bed must follow the procedure, with opiates administered for three days. The drain is removed on the fourth day, and the patient gets up. Elimination of the colloidal silver is rapid for the first few days. Chronic spermatocystitis transforms the seminal vesicles into foci of infection capable of harboring the infection for years.

UROGRAPHY

Swick [49] proposed sodium ortho-iodohippurate (hippuran), a halogen derivative of a compound normally found in the urine of man, as a medium for excretion urography. On the basis of its use in more than 200 cases, Swick has found the substance to be nontoxic, highly soluble and neutral in solution, and to yield satisfactory urograms. He stated that it possesses 38.8 per cent of iodine in stable organic union. Iodism has never been observed. Generalized warmth and, occasionally, nausea and vomiting of very transient duration have been the only reactions noted. Thrombosis at the site of injection has not been observed. The compound is well tolerated and may be recovered from the urine as the insoluble acid. Rabbits were found to tolerate from 2 to 2.5 Gm. of substance per kilogram of body weight, administered intravenously

49. Swick, Moses: Excretion Urography, J. A. M. A. **101**:1853 (Dec. 9) 1933.

in approximately 30 per cent concentration over a period of about ten minutes. Normally, from 90 to 95 per cent is excreted within eight hours after injection, from 60 to 66 per cent during the first hour and from 70 to 80 per cent during the first two hours. A solution of this substance remains unchanged in color or reaction after sterilization or on standing, and it may be prepared and distributed in sterile vials ready for use.

Swick stated that, for adults, a dose between 10 and 15 Gm. of this substance dissolved in distilled water, in approximately 50 per cent concentration, has been used. The injection should take about five minutes; the first exposure should be made about ten minutes later, and two subsequent exposures at twenty minute intervals. In cases of functional derangement, particularly in the presence of obstructive conditions, additional films are indicated to determine definitely the absence of visualization or the presence of late visualization.

Swick stated that oral administration in man has also yielded satisfactory results. Of 25 cases, in approximately 50 per cent good diagnostic urograms were obtainable from ninety to one hundred and thirty-five minutes after administration. From 10 to 15 Gm. of the substance dissolved in approximately 75 cc. of simple syrup was given by mouth. No reactions were noted. The only subjective sensation recorded was the salty, aromatic taste of the solution. Forty-five minutes after taking the solution, the patient is placed on the urographic table, a moderate degree of compression is applied, and exposures are made sixty, ninety, one hundred and twenty and one hundred and fifty minutes after ingestion of the substance. Swick stated further that, in order to obtain clearly defined and readable excretory urograms, it is important to apply a moderate degree of compression over the region of the urinary bladder by means of an air-inflated balloon held in place by the canvas sheet of the compressing apparatus.

Braasch [50] stated that excretory urography should and will be employed as a routine in the diagnosis of abdominal lesions. He said that its greatest handicap at present is in interpretation, but that this will become so standardized as to permit the method to be more generally employed. Its greatest value will be in determining the presence of stasis in the renal pelvis or ureter, in interpreting shadows in the upper part of the urinary tract and in giving fairly accurate estimates of renal function. Braasch also stated that it will always be an invaluable aid to the urologist in conditions in which cystoscopy and ureteral catheterization are impossible or inadvisable. It should be of help in determining the necessity for surgical treatment of renal ptosis. The data afforded

50. Braasch, W. F.: The Practical Application of Excretory (Intravenous) Urography, J. A. M. A. **101**:1848 (Dec. 9) 1933.

by this routine procedure should be complementary to other urologic data, and in only a limited field will it entirely replace former data.

Ravasini [51] stated that intravenous urography or, more correctly, urography by excretion of a shadow-giving or contrast substance, is an addition to the diagnostic resources in urology. Coincident with the use of the newer preparations, the inconveniences and accidents of the older ascending or retrograde method have been overcome. In the majority of cases, the newer methods give information not only as to pathologic changes in the kidney, but also as to any alteration in its functional capacity. The detailed observations of Ravasini were based on 1,000 of his cases in which intravenous urography was employed. In addition, an analysis is made of a large number of cases reported by other Italian and many foreign urologists. Ravasini has studied the behavior of the normal and pathologic kidney in eliminating the contrast medium, nephrography and the appearance and intensity of shadows and factors which show the relation between the intensity of the shadow and functional capacity of the kidney. He also studied the peristaltic movements of the renal pelvis and ureter and the manner in which intravenous urography yields valuable information as to both the anatomic and the physiologic condition of the ureter.

Ward [52] has had experience with pyelography by the descending route, or excretion urography, since 1929, and he has tested such chemical products as have generally been available for the purpose. Patients suffering from certain diseases are generally recognized as being unsuitable for investigation by this method. In the great majority of other cases, the method appears to be harmless, but on general principles it would seem wise not to use it repeatedly on the same person. It can be employed safely for children, and its value here is obvious, as also in many other conditions in which catheterization of the ureters is impossible. With very small infants, it is difficult to obtain shadows of sufficient density for diagnostic purposes. Ward stated that descending pyelography is a method of investigation that never fails to give valuable information when any disease of the kidneys and ureters exists. By means of this method, the health of these organs can often be established with great precision. It is also of use, although to a lesser extent, in the investigation of some vesical conditions. It does not replace ascending pyelography; the two are distinct and each has its own peculiar value. The former can almost always be employed; it gives information

51. Ravasini, Carlo: Pielografia per via discendente, Fifth Cong. Internat. Soc. Urol., 1933, p. 315.

52. Ward, R. O.: Pyelography by the Descending Route, Otherwise Known as Excretion Urography, or Intravenous Urography, Fifth Cong. Internat. Soc. Urol., 1933, p. 356.

of the greatest value concerning the physiologic condition of the kidneys and ureters and, in the majority of cases, depicts clearly their anatomic condition as well; but the latter reveals even better the anatomic details of renal cavities and has the advantage that specimens of urine from each ureter can also be collected. However, its employment is not always possible. Excretory urography makes it possible to study the activities of the kidneys and ureters while they are in an entirely undisturbed condition, and one which is physiologic except for the stimulus to secretion which occurs as a result of injection.

Ward stated that when partial stasis exists the most intense shadow usually develops after thirty minutes. Faint and varying shadows in the kidney and ureters which appear and fade away early indicate health. Dense shadows of constant form are not normal; they indicate a capacity to secrete, but a disorder in the conductive mechanism, and when late in appearance they give proof of impairment of the first of these two functions alone. A healthy ureter is never found filled with opaque fluid throughout its whole length at one time, the one exception to this rule being the ureters of the female during pregnancy. After his extensive study of this procedure, Ward stated that descending pyelography has given an even greater contribution to urology by the manner in which it makes visible the actions of the ureters in health, and that when the ureters are diseased it always reveals this condition; moreover, the results of operation on them and on the renal pelvis can be determined as was never before possible.

LYMPHOGRANULOMA INGUINALE

Cole [53] stated that lymphogranuloma inguinale is a distinct granulomatous entity involving the lymph nodes. After an incubation period of from one to several weeks, not necessarily accompanied by a primary sore, there results a chronic condition with the formation of buboes which eventually suppurate. The lymph nodes around the lower portion of the rectum may be involved, the inflammatory reaction often resulting in strictures of the rectum. Cole said that the cause of lymphogranuloma inguinale is a filtrable virus which can be transferred to several of the lower animals. He stated that a specific diagnostic cutaneous reaction (Frei reaction) has been evolved, the emulsion material from unbroken involved nodes being used as antigen. Cole stated that patients with buboes who are seen early respond comparatively well to surgical excision of the involved nodes or to intravenous injection of a solution of antimony and potassium tartrate. This disease, he says, is by no means rare in America.

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53. Cole, H. N.: Lymphogranuloma Inguinale, the Fourth Venereal Disease: Its Relation to Stricture of Rectum, J. A. M. A. **101**:1069 (Sept. 30) 1933.

CAUDAL ANESTHESIA

Campbell [54] stated that the results of caudal anesthesia in 83 boys parallel the observations of others in larger series of adults. In 67 (80.7 per cent) cases, anesthesia was satisfactory, which means that instrumentation was painless; in 7 (8.4 per cent), it was partially satisfactory, instrumentation having been performed with some discomfort. Campbell stated that the indications for, contraindications to and the administrative technic of caudal block are fundamentally alike for children and for adults. Among a great many children the employment of caudal block will eliminate general anesthesia. This is particularly desirable when the question of anesthesia delays or forbids the early establishment of the correct diagnosis and the institution of adequate treatment. In this group, the ease of administration and the results strongly recommend caudal block.

54. Campbell, M. F.: Caudal Anesthesia in Children, J. Urol. **30**:245 (Aug.) 1933.

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